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The Jackson Lecture.[1]

SOME VOYAGES CONNECTED WITH THE DISCOVERY OF AUSTRALIA: THEIR MEDICAL HISTORY.

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FIRST let me thank you for the kind thought that prompted you to institute an historical lecture in connexion with the Queensland Branch of the British Medical Association, and to associate it with my name. I appreciate very highly the honour which you have so done me. Secondly, I have to thank you for having accorded me the honour of giving the first lecture of the series.

It has not been an easy matter to select a subject. It was a fortnight or more after the date for this lecture had been fixed before I decided upon one. I have delivered many "historical" addresses in the last ten years, some of them in this hall, and I have written thousands of pages. Some of these pages you have heard; many of them have been delivered before historical societies in this State and in other States. The task of finding a subject upon which to address you should have been an easy one; but it seemed to me that the suitable theme for me to address you upon would be one in which there was a definite association of history with medicine. That consideration introduced a difficulty. At last, turning the matter over and over in my mind, I hit upon what I thought was the very thing. Thereafter, with pen in hand, I sat writing for two or three nights, till, as I judged, I had broken the back of my task. And then I made a discovery, quite by accident. I found that most of what I had written I had delivered before you on a previous occasion. The rest had been much better done for another Branc' of the British Medical Association by my friend, t' late Dr. Norman Dunlop, of Sydney. And so,

[1]Delivered at Brisbane, September 4, 1931.

preparing my subject, I have chosen a main historical path from which I have diverted along certain medical by-paths that have suggested themselves to me as I went on.

I exhibit a map of the world which I have prepared. It will help me to mix a little geography with the history. In doing that I may make myself clearer to you.

I shall try to sketch for you some of the incidents associated with the discovery of Australia. There are certain aspects of these incidents which, as medical men, you will be able to appreciate (dare I say it?) better than laymen. Let me take your minds back to the latter end of the thirteenth century.

We are apt to forget that Europe, Asia and Africa, until the Suez Canal was cut, were practically one great continent, and that it was possible to get from one end to the other of that continent by land. Up to the thirteenth century there had been no communication by sea between the extreme western and the extreme eastern shores of this huge piece of land. The chief journeys from the west to the east had been by means of caravans and such-like conveyances, from Genoa, Venice, Constantinople, or the ports on the shores of Asia Minor or Palestine. There were, in truth, comparatively few records even of such visits. (Hakluyt's "Principal Navigations, Voyages, Traffics and Discoveries of the English Nation," was published 1589-1600.)

Marco Polo (Thirteenth Century).

In 1260 two Venetians, Nicolo and Matteo Polo, set out to visit the East and before returning home in 1269 had even reached Cathay (or China). In 1271 they made a second journey, this time taking with them their son and nephew, Marco Polo, a youth of fifteen years. They sailed from Venice to Acre, and eventually, travelling by land, reached Cathay, or modern China. Here Marco Polo became the trusted councillor of Kublai Khan and remained in China for seventeen years or more. Anxious to get back to his homeland, Marco Polo made several attempts to get the necessary permission from the Khan, and, having got it, was entrusted coincidentally, with the job of conveying a young lady of high lineage from Cathay to Persia, of which country she was to marry the Khan, or ruler. A splendid squadron was fitted out, and Marco Polo, his father and uncle, embarked on one of the ships with the lovely seventeen year old princess and the three ambassadors, who had been sent for her from Persia.

So Marco Polo's return journey from China was made by sea as far as the Persian Gulf, following at first the coast of Siam. He came further on to the Malacca Strait, through which he passed to the Andaman Islands. Thence he sailed to the northern end of Ceylon, through Palk Strait and up the west coast of India into the Persian Gulf. And so by land to Trebizond, and by water to Constantinople and Venice.

The voyage from China occupied two years, and by the time Marco Polo and his companions reached Persia the Persian Khan, the intended bridegroom, was dead. The son slipped into his father's matrimonial shoes.

The expedition had lost six hundred men in that two years—nearly six men a week—between China and Persia. There is no record of the causes of these deaths, but we are justified in attributing them to scurvy, dysentery and malaria.

The history of Marco Polo's experiences and the description of the Asian countries, which he visited, as dictated by himself, fill about four hundred pages of a closely printed type. He and his father and uncle were perhaps the first Europeans to see the wonders of "The East," and they brought back great material wealth. Polo's story then inspired the adventurous spirits of his age to emulate his experience. The covetous eye of the West was henceforth upon the wealth of the East. It followed then, in course of time, that men should seek an easier way from West to East than that which the Polos had taken. The route by sea might be less difficult.

Henry the Navigator (Fifteenth Century).

Henry the Navigator (so called because of his interest for over fifty years in discoveries by sea), a Portuguese prince, third son of John the Great, became imbued early in the fifteenth century with the idea that there was a way to be found to the East by sailing south along the west coast of Africa and passing round the southern end of that Continent.

Henry took up his residence at Sagres in the south-west corner of Portugal and there set himself to study the sciences associated with navigation. Improvements in nautical instruments, such as the astrolabe and the compass, combined with charts which were no longer as haphazard as they had been, were the result. Portuguese charts began to record more accurately the journeys of Portuguese navigators. They were no longer mere guesses, but recorded what had actually been seen from Portuguese ships.

Henry now dispatched ship after ship under his most capable captains to explore southward along the west coast of Africa, hoping some day that one of them would find his way round the southern extremity of that Continent, and so up its eastern shores in the direction of India, and the still more remote Cathay.

In the map of the world which I show you, you will see recorded discoveries and re-discoveries for which Henry's navigators were responsible. Count among the re-discoveries Madeira, which had been involuntarily discovered in 1344 by a young Englishman, named Robert Machin, who, while eloping to France with another man's wife (who before being married against her will had plighted her troth to Machin), was blown thither by driving gales. The ship's company landed safely and made an encampment "on a flowery meadow in a sheltered valley." But the lady had been very seasick, and very depressed, before they reached the island, and when their ship broke from her anchorage and disappeared, her cup of woe o'erflowed. At the end of three days her troubled spirit departed and her body was buried. Whereupon Machin himself fell into the depths of despondency and neither ate nor spoke for five days. Then he, too, with touching fidelity, departed this life and was buried beside her in the woods.

Their companions then repaired the boat in which they had landed, sailed from the island, and were cast upon the Moroccan coast. Here they were thrown into prison, and found a companion in misfortune in a Spanish pilot, one Juan de Morales, to whom they told their story. This man was fortunate enough to secure his release, and eventually all that he learned from his fellow prisoners was communicated to Prince Henry of Portugal.

So Madeira, discovered in 1344 by Machin, was re-discovered by one of Henry's captains, and there in 1425 was established a Portuguese colony under Bartolomeo Perestrello. There was an especial importance in the appointment of Bartolomeo to this command because he afterwards (about 1478) became the father-in-law of Christopher Columbus. That great seaman learned much about Madeira from his mother-in-law, Donna Isabel Perestrello, whose husband, the aforementioned Bartolomeo, had died in 1457. She told him many stories of Madeira and its adjacent group of islands, and better still produced her husband's charts. Thus the knowledge of Columbus was increased and his ambitions were stimulated.

Porto Santo, an island neighbour of Madeira, had been the headquarters of Donna Isabel's husband. Thither, in 1479, Columbus took his bride, and there he lived for about three years. Moreover, in one of the expeditions which followed the death of Henry the Navigator and for which he was originally responsible, Columbus was in command of a small vessel in a fleet which went to Cape Coast Castle in the year 1481, under Admiral Azumbaga.

What he learned during his stay in Madeira and during that voyage only stimulated his curiosity as to what lands lay westwards across the Atlantic, and helped to formulate his plans to reach far Cathay by a voyage in that direction. He knew the world was round, but underestimated the distance between Europe and China.

But, for a while, let us return to Prince Henry of Portugal, his captains and their doings. They are but feebly recorded in the map which I exhibit. Adown the west coast of Africa, if you look closely, you will see printed at various levels the names of Portuguese commanders, each with a date. These figures mean that in the years thus recorded the particular captain whose name appears therewith reached that particular part of the coast. And as you look at the names, one after another, you will find that, as your examination takes you further and further south, the date recorded grows later. Thus it will be plain to you that as time went on Portuguese captains, at Henry's behest, kept on advancing down the west coast of Africa, till in 1461, one de Cintra, of Portugal, reached the neighbourhood of Sierra Leone. De Cintra was apparently the last navigator whom Prince Henry dispatched. His Royal Highness had died before de Cintra returned from Sierra Leone. In the forty-two years of Henry's work his navigators explored eighteen degrees of the African coast and had discovered, or re-discovered, the Azores, Madeira, Canary, and Cape de Verde Islands, and Portugal had more than begun to take first place as a naval power.

The great hope of Henry, as we know, had been that one day his ships would go far enough down the west coast of Africa to find a southern end to Africa, round which they could sail to India. If he knew and believed, as is most probable, the story of the voyage made by the Phœnicians in the sixth century B.C., which I am about to remind you of—then, I say, he must have been very sanguine indeed that final success would attend the endeavours of his men to pass round the southern end of Africa.

Herodotus states that in the sixth century Necho, a Phœnician ruler, King of Egypt, dispatched an expedition from the Red Sea, the vessels of which were to pass down the east coast of Africa, till at last they could round the Cape of Good Hope, and turning towards the north, eventually come back to Egypt, through the Mediterranean. They were three years accomplishing this voyage, and those concerned overcame the difficulties of food supply in a remarkable way. When autumn came they went ashore and sowed the land in whatever part of Libia (Africa) they happened to be sailing, and waited for the harvest. Then, having reaped the corn, they put to sea again. This story is sufficient to indicate that want of fresh food was known, even at that date, as a potent cause of the sailors' scourge, scurvy.

It is, I think you will agree, likely that during the voyage of these Phœnicians their staple diet was a cereal, and that most likely, it was wheat. Whichever cereal it was, it is certain that before consumption it had undergone no process of milling analogous to that which wheat undergoes in these days. It seems to be generally admitted that modern milling processes deprive our cereal flours of certain important body-building constituents, and even of material which assists the digestion of carbohydrates. Millers are not wholly to blame. The great "intelligent public" demands a flour that is perfectly white and it must have it. So the staple diet of children, women and men (note the order in which I put these classes) is continually deprived of some of its most important ingredients, and no one derives any advantage from this deprivation except the doctors and the dentists. Rightly or wrongly it has become, in certain quarters, a reproach to our profession. The public is not to blame, because, for the most part, "they know not what they do!"

There are those who tell us that it doesn't much matter, because what you have lost from your bread and your porridge you can make up with something else. That sort of advice, however, in my view, good as it may be, is not "good enough," and certainly will become, as time goes on, a greater reproach to us. For the poor, at any rate, a staple diet must be complete and cheap.

The history of the effects of this deprivation of the body-building ingredients belongs to modern days. In my own experience I can recollect the days when it was not uncommon to find middle-aged men and women with full sets of perfect natural teeth. To-day such a find is exceptional, and I believe that this deterioration is coincident with the so-called "improvement" in milling processes, which appear to have no object except to produce a meal as nearly white as possible.

Further evidence of the serious faults in civilized diet I have seen is a similar deterioration of the teeth in the members of various aboriginal races who have come under my notice. So soon as they give up the use of their native foods in favour of a European dietary, their teeth show the effects in caries and various other deficiency. Curiously, too, the magnificent "ivories" which they possess in their own countries are maintained without aid from the toothbrush. But this happy freedom from dental caries is not secure even in their own island homes when they give up native foods in favour of a cilivized diet.

Again, in my long professional experience I carry my mind back to the day when the word "appendicitis" was absent from the nomenclature of disease. (The incidence of this disease has kept pace, too, with the milling and with the deterioration in teeth). We are told that in old days the deaths from it were recorded under such heads as "General Peritonitis," "Perithyphlitis," "Psoas Abscess," *et cetera*, and that as diagnosis improved the cases recorded under these headings began to appear under that of "Appendicitis." The suggestion has been that there are in these days no more cases in proportion to population than there used to be. But if you search the records of hospitals of forty to sixty years ago you will find that such groups added together bear to the population of those days nothing approaching the gigantic proportion of appendicitis cases to the present day population. If further instances of the far-reaching effects of abandoning natural in favour of artificial foods and foods artificially prepared, are required, consider the enormous increase in necessary removals of tonsils for disease of those glands, which is often due to the condition of the teeth. I do not propose to attribute all cases of tonsillitis to deficient teeth, but you will certainly agree with me that most of those which are not caused by dental caries *et cetera*, are due to repeated catarrhs "caught" in the schools by the attending children, and often communicated to their parents, their cousins and their aunts. No preventive measures in regard to catarrhs in school children are in general use.

Again, as surely as anything can be, there is a close connexion between dental caries, gastric and duodenal ulcers, and cancers of the alimentary canal from the lips at its entrance to the rectum at its exit. Alongside the treatment by knife, by X rays, by radium, and all the newest of the new, the public must continue to be taught methods of prevention. That teaching should emphasize the need for a due supply of vitamins, body-builders, or whatever we like to call them. These are among the most important needs of gestant mothers, as well as of their offspring. Propaganda to impress the need for early treatment of cancer may be enough in regard to skin and external cancer. It is not enough in regard to cancers of the alimentary canal, which probably have no connexion at all with exposure of the human skin to the sun's rays. Directions for the prevention of this will be based on causes far less obvious than the sun's rays.

Finally, we are, I think, justified in assuming that the Phœnicians were sustained during this three years' voyage upon a staple diet derived from a grain, which had been only roughly ground. It was in fact a whole meal. I shall tell you presently of a voyage of between 6,000 and 7,000 miles, about 120 degrees of longitude, during which the only diet was for each man *per diem* eight to ten spoonfuls of a hominy made of maize. During that voyage at least one man recovered from a dropsy which was probably a species of beri-beri, and even gained strength in spite of the shortness of the supply.

After Henry's death in 1460 the Portuguese captains went on, under the instruction of their new ruler, each expedition creeping farther and farther south along the west coast of Africa. Their hopes were high, of course, when they found that at Cape Palmas it turned abruptly to the east; and when, near the island of Fernando Po it again turned south, they were correspondingly depressed. At last, however, in 1486, Bartholomew Diaz was blown south and east of the Cape of Good Hope, and as he was returning from his furthest southerly and eastern point he came upon the inlet now known as Algoa Bay, and touched therein at the modern Port Elizabeth, east of Cape Recipe.

At this point I would have you notice that the voyages of these men of Portugal as a rule followed the coasts pretty closely, and therefore permitted frequent landings for "refreshments." Apart from the actual foods so brought within their reach, there was the important factor of fresh water. (It was customary with some to burn out the inside of the casks in which water was carried, and this was especially necessary if they had previously contained oil. Later also it became the practice to "ventilate," or as we should say "aerate" the water by pumping air through it with a specially invented machine. But it is to be feared that this was too often only done when the water had already become "stinking.")

There is in the literature available to me and connected with these Portuguese voyages on the African coasts no mention of scurvy, and but little of disease of any kind. Illness at least did not present such serious obstacles as in later and longer voyages.

Eleven years after Diaz came the voyage of Vasco da Gama. This Portuguese captain rounded South Africa, and on Christmas Day arrived at Port Natal, to which he gave that name in reference to the date (December 25, 1497) when he arrived there.

After that da Gama passed farther up the east coast of Africa, visiting several Arabian colonies on the coast, till finally he reached Melindi. Thence, with the aid of Arabian pilots, he made his way to Calicut on the west coast of India.

By the time he returned to Portugal, da Gama had thus shown the way to India, and the extension of Portuguese dominion on the northern shores of the Indian Ocean rapidly followed. In a few years Portugal had the command of all the important straits at the entrances to that ocean from its various gulfs—Socotra, the Straits of Ormuz, the Straits of Malacca. Only Aden held out. Settlements were founded along the shores of the Arabian Sea and of India, especially at Goa and Calicut. Portugal defeated the Arabian fleets and became absolute

mistress of the west coasts of India. "The Portuguese route" to the Indies and the Spice Islands then became for many years the regular track to those countries for adventuring navigators.

Christopher Columbus (Fifteenth Century).

While Henry the Navigator and his successor were busy sending their men to find a way round the south of Africa to the East Indies, Christopher Columbus, who had served the Portuguese king as seaman and soldier, was turning over in his mind the prospects of finding a way to Cathay and the East Indies by sailing towards the west. His celebrated "first" voyage, which was the result of such cogitations, was made in 1492, or actually five years before da Gama made his successful voyage to India by what afterwards became the "Portuguese route" to the East. Columbus, indeed, did not get to the goal for which he was aiming. He, in his first voyage, actually got only to the Bahamas.

He left Palos on August 2, 1492. There was some delay over a mishap to one ship of his fleet, which necessitated putting into the Canary Islands for repairs, and eventually the ship did not get away from Gomera—the most westerly of those islands—until September 6. On October 11 he reached Watling Island, one of the Bahama Group, as it is now called, and gave it the name of San Salvador. It was originally known by the aboriginals as "Guanahani." The voyage had lasted just seven weeks from his last port of call. Time enough, as ship's diet went in those days, to lay the foundation for, if not to build, an attack of scurvy.

In the accounts of this voyage up to October 11 there is no mention of any sickness on board the fleet, so we are at liberty to infer that there was little or none up to that time.

Columbus, however, in reporting to his Spanish masters at a later date, informed their Royal Highnesses that soon after their arrival at San Salvador the greater part of his people had fallen suddenly ill. The freshly turned rank soil is supposed to have had a bad effect on the health of the garrison, and to have given rise to dangerous malarial vapours at night. Further he says:

> As we have seen among those who went by land to make discoveries, the greater part fall sick after returning. Some of them have even been obliged to turn back on the road.

And he goes on to say:

> Confiding in the mercy of God, Who in everything and for everything has guided us as far as here, these people will quickly become convalescent, as they are already doing. It is certain that if they had some fresh meat . . . all, with the aid of God, would very quickly be on foot.

The two most likely sources of this epidemic among his men were scurvy and malaria. Dysentery and yellow fever may have been in the picture. The scurvy might easily have been ripe to make its appearance after thirty-six to forty days of food deficient in nutritive value, such as the garrison had after leaving Gomera. Columbus speaks so hopefully about the prospects of a return to good health "if they had some fresh meat" that it would appear that the symptoms of some of his patients were those of scurvy, with which he would be likely to be quite familiar.

Through his messenger to their Highnesses he says as to the causation of their troubles:

> It has been said that the cause of the general sickness common to all is the change of water and air, because we see that it extends to all conditions, and few are in danger.

And he emphasises that

> These people be provided with the provisions to which they are accustomed in Spain, because neither they, nor others who may come now, will be able to serve their Highnesses if they are not well.

He suggests that that provision must continue until a supply is accumulated "from what shall be sowed and planted here." He then suggests the varieties of plants which are likely to afford the relief required. He emphasizes wheat and barley and vines. He reminds them that fruit must be waited for, promising also that with the wonderful soil of the Bahamas the products of Andalusia and Sicily would grow with great profusion. "No other land," says he, "on which the sun shines can appear better or more beautiful."

Columbus's idea that the sickness of his people was due to the turning over of new soil was one that lived till comparatively recently. It is curious how long it lasted. In these days, at any rate, most men of my age can say that they have turned over a good deal of new soil in their day, either with spade, fork, or plough, and that they have derived much pleasure from the results on the one hand, and no evil, in the way of sickness, on the other. Yet very different experiences seem to befall those who turn over new soil in mining camps, in waterworks camps *et cetera*. In such cases where more than two or three are gathered together in one camp there is invariably sickness, but due as we now know to faulty sanitation and faulty disposal of the sewage matter, household slops *et cetera*. And after all, Captain Henry Miller, the first commandant of the Moreton Bay Settlement and his immediate successors, 333 years after Columbus, had difficulties which were on all fours with those of the latter. That settlement was in the beginning nearly wrecked by malaria and dysentery, combined with rheumatism and ophthalmia; scurvy, too, of a mild kind was a trouble. This latter disease was soon overcome by the vegetables grown on the good arable land where the Botanical Gardens now are.

Columbus, at Watling Island, was not far from the home of yellow fever, nor from that island which once afforded a mortality of over 90% from yellow fever.

It seems obvious that the inclusion of vines by Columbus in his requisition was associated with a belief in the efficacy of wine for diseases due to deficiencies in diet. He bewails the fact that much of the wine which the fleet brought was wasted on the journey, and attributes this to the bad workmanship of the coopers who made casks for them in Saville. He frankly admits that "the greatest necessity we feel here at the present time is for wines, and it is what we desire most to have." "Although we have biscuit as well as wheat to last for a longer time, they also must be sent in reasonable quantity." He asks "for some salted meat—bacon, for instance—and for salt meat better than that we brought on this journey."

Presumably scurvy is a surer thing with bad salt meat, such as had been supplied to the fleet, than if it were good! I think that the fact was proved by Nansen. Fresh meat, of course, is better than salt, even if the latter does not "stink." But stinking meats, decayed whale, for instance, were constantly consumed by our blacks with no bad effect upon anything but their breath. It was never salted and often not cooked.

Describing the fresh meat that he would desire, he asks for lambs "and little ewe-lambs, more females than males, and some little yearling calves, male and female, and some he-asses and she-asses, and some mares for labour and breeding." And after all, the things which Columbus longed for when he reached Watling Island were pertty much the same as the things which were longed for by Governor Arthur Phillip soon after he landed on the shores of Port Jackson. Phillip had no opportunity of reading the history of this voyage of Columbus. One wonders, however, if a knowledge of these troubles of the Portuguese might not have made things better for the people of the First Fleet.

To add to all Columbus's difficulties, whatever form of fever it was that attacked his people, he himself suffered therefrom, and endured for some weeks all its pains and lassitudes.[1]

Columbus made three more voyages afterwards to and from the West Indies and its various islands. In one or more of them he went along the coast of South America from about Cayenne to Trinidad and along the coasts of the Isthmus of Panama. After that, men of various nations set to work to sail down into the higher southern latitudes along the coasts of South America. De Cabral went down to 20° in 1500 and in 1502 Americo Vespucci explored the coast down to 35° of latitude.

This was the position with regard to exploration of the eastern shores of South America when Magellan came to make his celebrated voyage through the Straits which bear his name. All the coast on the east and north of South America was discovered from Rio Grand to Panama.

Magellan (Sixteenth Century).

In 1519-21, as a natural sequence of the work of Columbus, came the conquest of Mexico by Cortes, and of Peru by Pizarro in 1531, and after that date Spanish settlements were made along the shores of both countries. Europeans then found themselves provided with a western route from Spain to the East Indies. It involved crossing the Atlantic to the neighbourhood of the Gulf of Mexico, crossing Panama to its western shores, and thence on the Pacific to the east, starting from Acapulco (Mexico) or Lima (Peru).

Ferdinand Magellan, a Portuguese soldier and seaman, who had given good service to his country, during which he had met that Francisco Serrana who had sailed in the north-west Pacific with a fleet of ships from Java, conceived the idea of finding his way from Europe to the Spice Islands by sailing round the lower end of South America. Serrano had seen many islands east of Java, and had finally settled in Ternate among the Moluccas. He wrote to Magellan of new lands to acquire in that direction. Magellan must have known that the east coasts of South America had already been visited by men from different nations, including Americo Vespucci, and by the year 1502 the coasts from Panama to the Rio del la Plata were known. But he was not aware of the existence of the straits which bear his name.

Magellan first made his proposals to the Portuguese king, who, as one might expect, rejected them. The Papal Bull of Alexander VI (May, 1493) which drew a meridian a hundred miles west of the Azores and then allotted to Spain all that was west of it and to Portugal all that was east of it, practically forbade the Portuguese king to encourage his people to sail towards the west in order to extend his dominions in that direction. So, for Magellan, nothing was left but to transfer his application from the Portuguese to the Spanish king (Charles V), and so, at length, his suggestions received the attention they deserved.

Commanding a fleet collected by the order of that king, Magellan sailed down the eastern coast of America and passed through the straits which bear his name in 1520. Afterwards he crossed the Pacific more or less in the north-western direction and reached the Philippine Islands.

Here is another long voyage made by the Spaniards with disastrous results to the crew of the ship. There is no time this evening for any long notice of the privations and serious mutinies which took place during the voyage under Magellan. They were only quelled by the determined Magellan shooting one mutineer, beheading another, and marooning still another. (I commend to your notice the story as told by Captain W. B. Whall in his "Romance of Navigations.")

Magellan reached the Ladrones, a group of islands in the north-west Pacific, so named by himself (the word "ladrones" meaning "thieves"). A few days here with refreshments of fresh vegetables, fruit and meat, did much for his crew. After passing to the Philippines, Magellan was treacherously slain by a native chief. His fleet, already depleted by the wreck of one ship and the desertion of another, was further reduced by the burning of one which bade fair to be derelict at no distant date.

The one remaining ship, the *Vittoria*, after Magellan's death sailed from the Philippines in December, 1521, commanded by Sebastian del Cano, and carrying a cargo of spices. She emerged into the Indian Ocean, and may have viewed from a distance some part of the western coast of Australia. Lower down in the Indian Ocean she discovered the islands of St. Paul and Amsterdam, before finally reaching San Lucar in Spain on September 6, 1522.

The *Vittoria* was thus the first ship to circumnavigate the Globe, as del Cano was the first commander to perform that feat. Of this honour Magellan was deprived by his untimely death.

And now the East Indies had been reached by both Spanish and Portuguese ships—the Spanish by

[1] The length of time at sea between Gomera and Watling Island in the voyage of Columbus constitutes the incubation stage of scurvy for that occasion. It is also a curious fact that in the histories of the voyages of Henry the Navigator's captains, of which I have spoken, there is not one word about scurvy. These voyages were all short ones, or if long, accomplished in short stages. It seems that it was not until voyages began to take six weeks to two months without sighting land, with no other meat than salt meat, and that often bad, that scurvy began to raise its evil head amongst ships' companies.

taking a westerly route from their homeland round the southern part of America and the Portuguese by an easterly route round South Africa. But at the price of what deprivations to Magellan and those under him can hardly be imagined, even after reading the following quotation from Pigafetta, who was a kind of official recorder to the fleet. It is perhaps right to quote him lest we forget the great patience and perseverance amid awful discomforts displayed by those old time navigators:

> We ate biscuit, but in truth it was biscuit no longer, but a powder full of worms, and in addition stinking with the urine of rats. So great was the want of food that we were forced to eat the hides with which the mainsail was covered. These hides, exposed to the sun, rain and wind, had become so hard that we were obliged first to soften them by putting them overboard for four or five days, after which we put them on the embers and ate them thus. We had also to use sawdust as food, and rats became such a delicacy that we paid half a ducat apiece for them.

Scurvy broke out with great virulence. On the arrival of the *Vittoria* in Spain only eighteen Europeans and four natives remained of those who had sailed in the fleet originally. In this voyage the want of opportunity for calling in at ports or islands for refreshments was the most potent factor in the causation of scurvy. While there were quite good opportunities in the Straits of Magellan for obtaining "refreshments," after leaving the Straits, Magellan never saw land till he reached Guam, an island in the Ladrones Group.

Magellan's voyage led to Spanish settlements in the Philippines. It was not until 1545 that the Spanish gave up the contest for supremacy in the Moluccas, and directing their attention to the north, began to form settlements. Thus Manilla was occupied by Spain in 1564, forty-four years after Magellan's voyage. Thereafter there was a regular service twice a year of ships between Manila and Acapulco in Mexico.

(From the end of the fifteenth century onwards the Portuguese route to the East Indies was used by the ships of England, Portugal and Holland, and perhaps to a less extent by those of Spain. After Magellan there were in constant use two or three routes for Spaniards into the Pacific and towards the east. One, of course, passed from the Atlantic to the Pacific through Magellan Straits, and in the latter part of the sixteenth century, if not before, there was for Spaniards also the route across the Isthmus of Panama, across America to its western shores, and thence either from Mexico or Peru across the Pacific to the Philippines.)

The route across the Pacific to Manila from the Spanish possessions was chiefly from Acapulco or Lima, and roughly speaking in latitudes between the tenth and twentieth degrees on either side of the Equator, more usually on its northern side. In returning from the Philippines to Acapulco it was the custom to seek higher latitudes than those followed in the voyage from the east to the west. This in accordance with prevailing winds.

Drake and Cavendish (Sixteenth Century).

By the middle of the sixteenth century the Spanish settlements on the western coast of America offered certain temptations to such adventurous commanders as Drake and Cavendish. To harass such settlements would serve more than one purpose: (i) It would afford the pleasure of singeing the Spanish beard, for the Spaniard was rather a nasty bully at this time. (ii) To deprive the Spanish king of valuable cargoes now passing every year from the Philippines to New Spain was to deplete the Spaniard's purse and reduce his capacity for building ships of war and collecting armaments. (iii) If the second purpose were fulfilled, it followed that the English purse would be filled—a matter which would please every English sailor who survived the expedition to share in the amounts realized by the disposal of loot. (iv) And moreover, as Queen Elizabeth, the English sovereign, was always a participator in the division of loot, there was a certainty of ingratiating Her Majesty. Such certainty in Drake's case led to her knighting him instead of hanging him, as Phillip II of Spain was demanding of her even before Drake got home.

These, then, were the chief reasons which, in the latter part of the sixteenth century, prompted such voyages as those undertaken by Drake (1577) and Cavendish (1586). The Armada came in 1588.

The voyages of Drake and Cavendish were not primarily voyages of discovery. Yet Drake discovered that Tierra del Fuego was not, as had been suggested, a portion of that Terra Australis—that great southern continent, the existence or non-existence of which was for so long a matter of dispute. After leaving the Straits of Magellan, fierce west winds blew him back south and east of Cape Horn, and thus enabled him to settle this point. He sailed up the American coast to Vancouver. He had some idea of finding his way back to England by some passage between Asia and America. His proceedings are not for us this evening.

Cavendish was a great seaman and a successful looter. Both he and Drake were *personæ gratæ* to Elizabeth, who knighted Drake.

Other Voyages in the Sixteenth Century.

During the latter half of the sixteenth century some very interesting voyages of discovery in a westerly direction were made from the ports of Mexico and Peru. None were more so than those of Mendana and Gallego (1567), Mendana and de Quiros (1595), and de Quiros and Torres (1605).

In 1567 Mendana, with Gallego as pilot, sailed from Lima and reached the Solomon Islands. Landing on the island of Ysabel in this group they examined several of the neighbouring islands during a period of some months. On his return to Lima Mendana made certain representations as to the discovery of gold, and as to other advantages to be derived from the formation of a settlement in the Solomon Islands. Accordingly, in 1595, nearly thirty years after his first voyage, he was entrusted with the command of an expedition to sail westwards for that purpose. His pilot on this occasion was de Quiros, and the fleet carried men, women and children—married couples, and single people, some of whom were married on the way. There were on board members of the Spanish priesthood who were authorized to perform marriage ceremonies and other religious functions.

In an age when for want of chronometers the longitudes taken by the mariners of the time were necessarily unreliable—practically never correct—it need not surprise us that Mendana failed to find the Solomon Islands. This time, however, he found the Santa Cruz Group (some five or six degrees east of Ysabel Island, and a little south). On the principal island he attempted to place his settlement. The project was attended by disasters of many kinds. Not the least of these was the death of Mendana himself. The command of the expedition then devolved upon the lady, his wife, who had accompanied him from Lima. She, with such pilotage as de Quiros gave her, sailed the fleet to Manila. De Quiros, after his return home, found his way again to Lima, always hankering to make a further attempt to find the Solomons.

In 1606 he commanded a new fleet with Luis de Vaes Torres as pilot, and went off to found a settlement in the Solomons. Here again the uncertainty of longitudes spoiled de Quiros's results. This time the fleet failed to find either the Solomons or the Santa Cruz Group, but eventually, *faute de mieux*, an attempt was made to place the settlement on one of the New Hebrides Group. To this island de Quiros gave the name Tierra Austrialia Espiritu Santo, by which it is known to this day, though more often it is spoken of as "Santo," for short. (The letter "i"—the sixth in the word "Austrialia"—may seem out of place. de Quiros introduced it as a compliment to the Spanish king, whose dynastic origin was from the royal house of Austria.)

From the Australian point of view the voyage of de Quiros and Torres had a special importance. They were instructed, on leaving Santa Cruz or the Solomons, to sail in a south-west direction till they reached 21° of south latitude in search of what land they might find, or a supposed Terra Australis. Having reached that latitude they were to sail north as far as 4°, and so round the already partially explored northern coast of New Guinea.

For some time before leaving Espiritu Santo the mariners who composed de Quiros's ship's company had displayed a mutinous spirit. After leaving the island one night, they mutinied in hard earnest and compelled their commander to sail back to the American coast. Torres, on the other hand, endeavoured to carry out the whole of the programme ordered, and accordingly sailed south-west as far as 21° south. From that position he turned his ship towards the north and sailed till he reached the south coast of Basilisk Island, off the east end of New Guinea. Hereabouts he examined and named islands around Millen Bay. Further west he spent some time examining Orangeries Bay, so named at a much later date by Bougainville, and finally he passed through the Straits which bear his name. Prado, who was upon Torres's ship and was something of a cartographer, wrote a journal which has only recently been found, and has upset the theory that in passing through the straits, Torres went near enough to Australia to get sight of Cape York. He was no further south than Banks Island, which lies about midway between New Guinea and Cape York, and though on the east coast he must have been near the Barrier Reef, he has no claim as a discoverer of Australia.

The Dutchmen in the East (Sixteenth and Seventeenth Centuries).

It was in the last decades of the sixteenth century and the first two of the seventeenth that Dutch power in the East Indies grew to important dimensions. We have already taken an historical trip into the margin of the seventeenth century of de Quiros's and Torres's Spanish ships. We may now fairly continue it further into that century—chiefly, to begin with, on Dutch vessels.

In November, 1605, the Dutch East India Company sent from Bantam a ship, the *Duyphen*, to look for a land said to exist south of their East Indian possessions. Eastwards she passed to the Key Islands and the Aru Islands, and reaching the south coast of New Guinea passed in a southerly direction into the Gulf of Carpentaria, till in 13·75° latitude she turned homewards opposite the Cape Keerweer of modern maps (the name of which, being interpreted, is Cape Turn-again). This is so far the first recorded sighting of Australia by Europeans. The *Duyphen* sailed back to Banda by June, 1606. The captain of the *Duyphen* had nothing good to say about that part of Australia which had come under his observation. There was nothing therein which promised any hope of profitable trade. The native inhabitants were ill-favoured, of low intelligence and hostile, so hostile that they killed some of the crew of the *Duyphen*. The land itself was, as they saw it, no less ill-favoured than its inhabitants.

The *Duyphen's* people did not recognize the opening between New Guinea and Australia as a strait, though it must have looked like one.

Dutchmen were looking for opportunities of commerce. There was little chance of their electing to send a settlement to that part of Australia while they were so near to the fertile Moluccas, which were full of such opportunities.

In 1611 an important change took place in the route ordinarily selected by ships bound from Western Europe to the Far East. The Portuguese route occupied twelve or even eighteen months. A Dutch captain, Hendrik Brouwer, proposed in 1611 that, instead of sailing north immediately after rounding the Cape of South Africa, ships should be kept on a due east course for a distance of about 4,000 miles. Having thus reached the longitude of Java the course should be altered to a northerly one. Thus, he declared, vessels would keep a wind coming more or less astern, and would make their passage quicker. They were always delayed by "doldrums" in the Indian Ocean when they took the Portuguese route. He finally showed that instead of taking twelve, fifteen or even eighteen months over their journey to Java they could do it in six months. Brouwer's route then became the ordinary one to Java, especially for Dutch ships.

You will recognize that the inevitable result of the adoption of this route would be that some captain would overshoot the mark at four thousand miles and would come upon the Australian coasts from the Cape. It must be remembered in this connexion

that in those days accurate estimate of longitude was impossible owing to the want of chronometers. A list of ships or navigators who thus overshot the mark would include among others:

1. 1627—Peter Nuyts—in the *Golden Leopard*, who passed along the south coast of Australia to Fowler's Bay.
2. 1622—The *Leeuwin* ship—C. Leeuwin.
3. 1619—D'Edels
   „ —Houtman
4. 1616—Dirk Hartog
5. 1628—De Witt

All came to the West Australian coasts in this way.

As to Australia's more northern shores, the *Arnhem* and the *Pera*, two yachts, were sent in 1623 from Amboyna by Governor Herman van Speult, of that Island, to follow the course of the *Duyphen* and proceed beyond Cape Keerweer. They were commanded by Carstens. The *Pera* went down to 17° 8′ along the west coast of Cape York, and went back along the New Guinea coast. The *Arnhem* separated from the *Pera* in the Gulf of Carpentaria, and returned along its west shore, since called Arnhem Land.

Before 1644, when Tasman's visit was made to our western coasts, they had been for the most past seen by other Dutch commanders. Tasman passed along the Australian coasts in 1644, found the Northern Territory, and later examined the shores of the Gulf of Carpentaria. It may be said that the adoption of Brouwer's route (or the Dutch route) to Java, after 1611, was indirectly responsible for the discovery of the major portion of Australia's western shores, and that in less than thirty years and by Dutchmen.

Dampier (Seventeenth Century).

Though Dutchmen earned the title of "discoverers" of the western shores of our continent because they were the first European commanders to see them, part of the West Australian coast was, in 1686 and 1699, intimately examined by that truly great and extraordinary Englishman, William Dampier.

You may say that the honour of being the first Europeans to see the shores of Australia on its north and west side from Cape Keerweer to Cape Leeuwin belongs to Dutchmen. Also the southern shores from Cape Leeuwin to the head of the Great Australian Bight. That is not to say that they saw and surveyed every mile of it, however. For the intimate survey of those shores in the north and west we have to thank Dampier and Reed in the *Cygnet*, in 1686, Dampier in the *Roebuck*, in 1699, Flinders in the *Investigator*, 1802 and 1803, and P. P. King in 1817 and onwards (two circumnavigations of our continent in the *Mermaid* and one in the *Bathurst*). It is an extraordinary thing that almost one never hears any talk of King's achievements. I commend to your notice Captain (and later Admiral) Phillip Parker King's book "The Australian Coasts." As things are, it is too common to find that even some of those who teach history in schools have no acquaintance with the magnitude of his work, and scarcely know his name. Captain Wickham and Stokes in the *Beagle* examined the shores omitted by those whom I have mentioned.

In 1686 the *Cygnet*, buccaneer ship, under Captain Swan—a capable seaman, and a very fat man, left the Galápagos Islands, off Mexico, for Manila, with William Dampier on board. Dampier was at first, at any rate, not an unwilling passenger. The intention of the buccaneer crew on reaching the Philippine waters was to lie in wait for the treasure ship which twice a year left Manila for Acapulco. Swan's desire was to get to England. Sick of buccaneering and his crazy crowd of ruffians, he was for throwing himself on the King's mercy, in an endeavour to rehabilitate his conscience. Dampier himself was glad of the chance of seeing a part of the world's seas which were new to him, to wit some of the latitudes above the Equator in the Pacific. Before starting on this journey of six thousand miles by sea, a stocktaking took place. They had less than sixty days' provisions "at a little more than half a pint of Maiz a day for each man, and no other provisions, except three meals of salted Jew fish." Even that was not to be relied upon because the multitude of "rats on board could not be hindered from eating part of our Maiz!"

They reckoned on a journey of 6,900 or 7,200 miles, nearly one hundred degrees of longitude. Guam, in the Ladrones, was the first place they could touch at. Dampier, a few days before starting from the Galápagos Islands, had cured himself of a dropsy, "a distemper from which many of their men had died," by a method which he thus describes:

> So here I was laid and covered all but my head in the hot sand. I endured it near half an hour, and then was taken out and laid to sweat in a tent. I did sweat exceedingly while I was in the sand, and I do believe it did me much good, for I grew well soon after.

What was the dropsy? Beri-beri or what?

Half a pint of maize *per diem*! Eight spoonfuls of boiled maize once a day! Later, ten spoonfuls. This was to be the menu during a voyage of six or seven thousand miles, which occupied 150 men and two ships for over seven weeks.

Dampier says:

> I do believe that this short allowance did me a great deal of good, though others were weakened by it, for I found my strength increased and my dropsy wore off. Yet I drank three times every twenty-four hours.

Many of the men "did not drink in nine or ten days." One of the men "did not drink in seventeen days' time, yet he made water every day, more or less." During the voyage they "did not see one fish nor any sort of fowl."

Nearing Guam the men were mutinous, planned to kill and eat Swan and Dampier, when other victuals were at an end. Swan first. When they came in sight of land this intended mutiny was lost sight of. Swan said—"Ah! Dampier! You would have made a poor meal!"

Now that story is surely an argument for the vitamins of maize, and perhaps there is promise of a grain of comfort for the dropsical in a "starvation diet" of eight to ten spoonfuls of boiled maize *per diem*—with a moderate amount of water to drink!

At the island of Mindanao in the Philippines, Dampier made, for him, another important medical discovery. The Mindanayans commonly:

Ease themselves and make water in the river. For that reason you shall always see abundance of people of both sexes in the river. Both men and women take great pleasure in swimming and washing themselves, being bred to it from their infancy. I do believe it is very wholesome to wash mornings and evenings in these hot countries, at least three or four days in the week.

He tried this treatment personally and found it very refreshing and comfortable. That it was good for fluxes "to wash and stand in the river mornings and evenings" he proved in his own person, and was quickly cured. Here is a new (or perhaps an old) cure for dysentery!

At Mindanao the *Cygnet's* buccaneers deserted Swan and sailed away, leaving that unpopular commander behind. The course of the *Cygnet* was eventually north and west of Timor till she emerged into the Indian Ocean. Dampier was a somewhat unwilling passenger with this crew of rascals, commanded by a new captain, whose name was Read. Nevertheless he was doubtless responsible for the course which took them to the west coast of Australia.

After that they sailed to the Nicobar Islands, where Dampier left, or was left by them. What to me was his most fascinating exploit occurred after this, when he sailed from Nicobar to Achin with companions in an outriggered canoe. It was a rough trip even for this experienced seaman and hardened sinner, and he wrote of it:

I made very sad reflections on my former life, and looked back with Horrour and Detestation on Actions which before I disliked, but now I trembled at the remembrance of. I had long before this repented me of that roving course of life, but never with such concern as now.

He "once more desired God's assistance" and was not disappointed in his hopes.

This great Englishman, William Dampier, was also concerned in another exploration of our western coasts. In brief the story of his voyage in the *Roebuck* can be told as follows: Sailing from England in 1799 in command of his crazy ship he passed islands off the west coast of Africa, on some of which he landed for refreshments. Thence he made Bahia on the coast of South America. From Bahia he sailed past the Cape of Good Hope across the Indian Ocean, and came on August 6 to the Australian coast at Shark's Bay, to which he gave that name. From that bay he worked his ship up the coast as far as Roebuck Bay, on the shores of which is now the modern town of Broome. He had some intercourse with natives while he was on shore searching for water, and on one occasion had to shoot one of them to prevent one of his crew from being killed. Unable to find water by digging on the shores of Shark's Bay the *Roebuck's* people were, however, "very well refreshed with raccoons, turtle, shark and other fish, and some fowls."

Dampier subsequently went on a northerly course alongshore, looking for a place favourable to the finding of good water. The islands known to-day as Dampier's Archipelago were seen and examined—again without finding water.

Dampier had a very poor opinion of Australian blacks—"the lowest and dirtiest of all"—and his experience of human kind was gained from all over the world. And yet that same "low and dirty" Australian aboriginal even to this day can live in the bush where the intelligent white man would starve. Many a white man in the past history of Australia has owed his life to the bush craft of a blackfellow.

Finally Dampier dismissed the Australian coast with these words:

And thus having ranged about a considerable time, upon this coast, without finding any good fresh water or any convenient place to clean the ship, and it being moreover the height of the dry season and my men growing scorbutick for want of refreshments, so that I had little incouragement to search further; I resolved to leave this coast, and accordingly in the beginning of September set sail for Timor.

After leaving Timor he sailed in a north-easterly direction, passed along parallel with the northern shores of New Guinea and round the east shore of New Ireland and south of New Britain, and between that island and New Guinea—along the shore of which he passed to the Moluccas and Batavia.

On her way home the *Roebuck* sank at Ascension Island. Her crazy timbers sprang a leak and would carry her no longer.

Bougainville et Alii (Seventeenth and Eighteenth Centuries).

For us, who are Australians, the most important voyage made into the Pacific and from the East was that of Cook. Many voyages which crossed the Pacific from the west were made before his in the seventeenth and eighteenth centuries. They, in addition to being forerunners of his great journey into the Pacific, contributed substantially to the knowledge of the great navigator, and so enabled him to formulate plans which under his direction were attended by success.

The following is a list of some of the most important of these voyages:

Le Maire and Schouten, 1615 (named St. of Le Maire and C. Horn).
Dampier in the *St. George*, 1702.
Woodes Rogers and Dampier, 1708.
Roggeween, 1722.
Anson, 1741.
Byron and Mouat, 1764-1766.
Wallis and Cartaret, 1766-1768.
Bougainville—a French voyager.

Thus many islands in the east and middle Pacific were discovered and named. With the exception of Bougainville, none of these men came near Australia. Bougainville visited Tahiti and the Navigator Group. He afterwards rediscovered the New Hebrides and then passed in an easterly direction in 15° S., till he came close to and saw the outside of the Barrier Reef. Thence he sailed north to the south coast of New Guinea along which he passed to the East.

Bougainville's next important discovery, while pursuing his course to the East, was the rediscovery of the Solomons. He had left Tahiti on April 16, 1768. On May 5, while among the Navigator Group, scarcity of water was compelling to imprudent navigation at night. (The custom in these voyages of discovery was to "bring to" at nights.)

Bougainville wrote:

The scurvy in the meanwhile made its appearance. A great part of the crew and almost all the officers had their gums affected, and the mouth inflamed with it. We had no refreshments left, except for the sick, and it is difficult to eat nothing but salt flesh and dried pulse.

At the same time there appeared in both ships several venereal complaints contracted at Tahiti. They had all the symptoms known in Europe. Bougainville had brought away from Tahiti a certain native. Him he ordered to be examined. He "was quite ruined by it."

The fact that venereal disease was rife in Tahiti during Bougainville's visit in 1768 effectually disposes of the charge that Cook's voyage in the *Endeavour* was responsible for its introduction, but does not place the responsibility.

The question as to which visitor was responsible for introducing syphilis or any venereal disease to the Society Islands is a difficult one to settle at this date. The following is a list of visitors to those islands, prior to Cook:

1765 Byron.
1767 Wallis.
1767 Cartaret.
1768 Bougainville.

Byron called at two islands, which he called King George's Island in June, 1765, and his people found there the carved head of a rudder which had manifestly belonged to a Dutch longboat. They found other evidence of the visit of a Dutch ship to these parts—a piece of hammered iron, a piece of brass, and some small iron tools, and the remains of an adze—worn away almost to nothing. This last indicated that the visit had been made long ago, possibly when the ancestors of the then living natives were in existence. There is no mention in Byron's journal of any venereal disease.

Wallis left Magellan on April 12, 1767. By April 29 many of his men were down with "colds and fevers" because the upperworks of the vessel were open and their clothes wet. On April 27 the weather was moderate and fair, and their clothes were dried. The sick were served with "salop and wheat boiled in portable soup, and all the ship's company had as much vinegar and mustard as they could use. Portable soup was also constantly boiled in their pease and oatmeal." By May 14 this ship's people were "mending apace," but again on May 22 Wallis made the following note:

> The people who had been recovering from colds and fevers now began to fall down in the scurvy, upon which (by order of the surgeon) wine was served to them: wort was also made for them of malt, and each man had a half pint of pickled cabbage every day.

One would have supposed that upon such a menu scurvy would have received a check. It is to be noted that the captain does not say whether the men who had been recovering from colds and fevers, and who were evidently the first to fall down with scurvy, had been able to consume the diet prescribed. Possibly the poor appetite of their convalescence had something to do with it.

But with all this care and attention the men grew pale and sickly and began to fall down very fast in the scurvy. Their quarters and their clothes were "kept perfectly clean," and the water they drank was "rendered wholesome by ventilation" and "every part of the 'tween decks was frequently washed with vinegar."

They were now among the Society Islands, of which group they saw and named three or four, and reached Otaheite (Tahiti) or King George the Third's Island, as Wallis named it, on June 19, 1767. (Two years after Byron in 1765 had visited two islands a little north of Tahiti and called them King George's Islands.)

After some preliminary skirmishes with the natives Wallis's ship was able to land her sick. Fruit and fresh provisions were served—fresh pork, fowls, fruit—and by June 28 all were "fresh and healthy"; they had fared sumptuously. (The fish supply, however, was disappointing to a degree. None was caught by either net or trawl.)

Those ships which came through Magellan Straits usually escaped scurvy till some weeks after leaving Cape Pillar at its western end; while those which came round Cape Horn usually were badly affected with that disease before getting into the Pacific. The reason for this difference is, of course, that there were opportunities of good "refreshments," and good water in the Straits. The length of time between the ship leaving the Straits of Magellan and the first appearance of scurvy depended, of course, on the condition of the crew in regard to that disease before entering the Strait, and the length of time they remained in it enjoying its good food and water. And of course the dietary with which the ship had been equipped before leaving her home port was a factor. If that were good, the freedom from scurvy was longer. Yet Cook's was the only ship among the many which practically escaped scurvy, though he came round the Horn.

With regard to venereal diseases at the Society Islands: Wallis expressed his certainty, on good grounds, that none of his crew, in spite of "free commerce" with the women of Tahiti, suffered from any venereal disease. Cook found that the disease was present when he came to the island, less than two years later. Between the visits of Wallis and Cook, however, the French ships *Boudense* and *Étoile*, commanded by Bougainville, and bent on a voyage of discovery, had come to Tahiti and had stayed there less than a fortnight. Within a month after the French ships left Tahiti several forms of venereal disease attacked Bougainville's crew.

It was not till 1786 that John Hunter, the great London surgeon, published his treatise on venereal disease, in which he related his experiences, based on the experimental inoculation with syphilis of himself and five students. That indeed made the differential diagnosis of syphilis a much easier matter than before, but this knowledge was not available to Bougainville, or Wallis, or Cook. None of these men even distinguished between gonorrhœa and syphilis.

The most reasonable explanation I have ever read of the disappearance of the so-called "leper" lazarettes which once studded England I found in an article by a medical man in an old book—"The Transactions of some Philosophical Society"—if my memory serves me correctly, published about 1804. It was simply that, after Hunter made easier the diagnosis of syphilis, that disease was no longer mistaken for leprosy. The lazarettes probably con-

tained almost no lepers. The people segregated in them were syphilitics.

Other Australian Discoverers, 1770-1802.

Up to the present in this address I have not touched at all on the exploration of the southern shores of Australia other than to mention the voyage of the Dutchman, Nuyts, which took him as far as the head of the Australian Bight in the year 1627.

Beyond that point as far as Encounter Bay Flinders (1802) was the first to examine the shores during his voyage in the *Investigator*. He had come last from the Cape of Good Hope, well equipped against scurvy, and on his arrival in Sydney a few weeks after leaving Encounter Bay his crew was in good health. The ship's company were all on deck coming into harbour and all well. Their fresh colour reminded onlookers of England. Apart from a proper diet, "a strict attention to cleanliness . . . a free circulation of air in the messing and sleeping places had been the essential parts" of the arrangements.

Beyond Encounter Bay Baudin (1802), the French explorer, in the *Géographe*, was responsible for the discovery of the shore between Encounter Bay and the South Australian border. Scurvy had assailed his crew after many months at sea, so that he was obliged to make for Port Jackson, through the heads of which he was unable to take his ship, for want of men fit to do the work. Here the men of the *Investigator* came to the rescue, were sent down to the French ship and brought her into Port. (June 18, 1802.)

British hospitality successfully stood the test of caring for a crew of sick men belonging to a hostile nation. Twenty-three men were taken into hospital and given the best that was to be had of everything. That "everything" compelled the slaughter of beef which the people of the English settlement could then only afford by pinching themselves, though the French exchanged good salt meat for it.

As director of such hospitalities, Governor Philip Gidley King left nothing to be desired. Baudin sailed on the *Géographe* on November 22, 1802, with a healthy crew. With him went the *Naturaliste*, the other ship of the French fleet, and a new ship, built principally of casuarine (coast she-oak) in Port Jackson, of 20 tons burden, and bearing the same name as her timber. She was sold to Baudin by a private owner with King's consent. The *Naturaliste* had also been disabled by scurvy and, like the *Géographe*, her crew was well cared for by King. After some further survey the *Naturaliste* was sent to the Isle of France *en route* for Europe, carrying specimens already collected. War had broken out and she was captured by the English but released almost at once. Baudin with his two remaining ships discovered Geographe Bay in Western Australia. He died in the Isle of France.

The kindness shown to Baudin by King while in Port Jackson was in strong contrast to the treatment of Flinders by Governor de Caen of the Isle of France in the following year.

James Grant. From Cape Banks to Westernport the shores of Australia were first seen in 1800 by Lieutenant Grant in the *Lady Nelson*, a sixty ton brig, with a two feet nine inch free board when loaded. She had sailed from England *viâ* the Cape of Good Hope with a crew of fifteen, all in excellent health when she reached Sydney.

George Bass, a surgeon, in the matter of discovery was responsible (1795) for the south coast of Australia from Westernport to Cape Hicks (now Point Everard). His work was done in a whale boat from Port Jackson. The men of his boat's crew were all in good health when he returned from Westernport to Port Jackson.

Lieutenant James Cook in 1770 was responsible for the discovery of the whole of the east coast of Australia, from Cape Hicks to Cape York. At this juncture I shall only notice his voyage, so well known to you all, by making a few remarks on the experiences of his ship's company at and after he left Batavia.

Conclusion.

I conclude with a final note on the mortality in the ship's company aboard Cook's *Endeavour* which arose in consequence of his visit to Batavia.

Up to the time of his arrival in that port he had lost seven men. Of these one had died of tuberculosis; two were frozen to death in Tierra del Fuego; one died of alcoholic poisoning and three were drowned. (Note that there were no deaths from scurvy, malaria or dysentery.) While on the coast of Australia two had shown symptoms of scurvy—Mr. Green and Tupia.

The *Endeavour* came into the Batavia Road on October 10, 1770, with her people so healthy that Banks wrote of them: "Our people . . . might truly be called rosy and plump. For we had not a sick man among us." The Batavians themselves "were pale as spectres."

The *Endeavour* left Batavia to water at Prince's Island in the Straits of Sunda on Christmas Day, 1770, *minus* seven of her people who had died since reaching Java. Forty of them had been sick with fever, with dysentery, or both. At Prince's Island water was taken aboard. Between the island and the Cape of Good Hope twenty-three persons died of "the flux" and were put overboard. It is idle to speculate whether the Batavian water or that which was shipped at Prince's Island was responsible for this disastrous outbreak. The casks used for the Prince's Island water had previously contained Batavian water, and so were doubtless infected. The total number of deaths before reaching the Cape of Good Hope on March 15, 1771, was thirty. Banks had a fever from which he nearly died, but was cured by bark. Obviously, therefore, it was malaria. Others were similarly affected. The water supply was treated with lime to purify it, and the strictest attention was paid to the cleanliness of the ship.

Nothing had seemed of much avail till the trade wind had been picked up, when the condition of many of the invalids improved. The surgeon was inclined to give credit for this to the breeze, but probably the cleansing of the water with lime was potent in their improvement. Still, there were twenty-eight sick men to go ashore at the Cape.

As to the cause of all this sickness, Batavia had an ill-omened reputation, and it has been estimated that from 1735 to 1755 no less than 1,000,000 deaths took place, chiefly from malarial fever and dysentery. (Kitson's "Life of Captain Cook.") Its canals, some with fresh water in them, and some with salt, were a

sufficient harbour for mosquitoes to explain the malaria. A contaminated water supply in Batavia and probably also in Prince's Island, and elsewhere, sufficiently explains the dysentery.

Batavian health matters are better to-day than they once were, though the climate leaves much to be desired. Forty or fifty years ago, when British-India boats were bringing immigrants to Queensland —four hundred a fortnight—they brought us neither malaria nor dysentery among their people. This, however, was probably due to the fact that immigrants were not allowed ashore. Permission to land was given only to first-class paying passengers. The anchorage at the wharf, which has been described as "a hell of a hole" was infested with mosquitoes, so that for an ordinary man sleep was impossible. It was not used on the outward passage by British-India ships. They remained at anchor in the roads.

Malarial fever is endemic in Java—and occasionally becomes epidemic among the native populations, and with huge mortality among them. Dysentery, both of the amœbic and of the bacillary forms, has still to be guarded against, but is less in evidence owing to greater supervision of the water supply.

Since 1911 there has been instituted by the Government a Public Health service. This has resulted in the establishment of a medical university. Native doctors are trained therein, passing through various grades: first as assistant nurse (men and women), next the individual passes into the laboratory for training, and finally becomes assistant surgeon. Further training is available both in the medical and dental professions as well as for chemists' assistants, analysts, and health inspectors, vaccinators *et cetera*.

So to-day, under enlightened Dutch medical supervision, the belief of the ignorant natives "that the devil whom they call Satan is the cause of all sickness and adversity, so that when they are sick or in distress they consecrate meat, money, and other things to him," is no longer permitted to operate to the detriment of the people of and the visitors to Batavia, whether the latter be traders, tourists or sailors. An epidemic of malaria or dysentery in any part of the country around Batavia now is quickly followed by exhaustive enquiry and control by the very competent officers of a Tropical Medical Institute. It is no longer possible for a dead cow to float in the canals of Batavia for a week at a time, or till a flood carries it off (Banks). The good work has been made possible by the discoveries relating to mosquito transmission of disease, which were made by Ross and others during my life time.

OBSERVATIONS ON THE CERTAINLY LETHAL DOSE OF THE VENOM OF THE COMMON BROWN SNAKE (DEMANSIA TEXTILIS) FOR THE COMMON LABORATORY ANIMALS.[1]

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The common brown snake, *Demansia textilis*, though widely distributed in Australia (occurring in Western and South Australia, Victoria, New South Wales and Queensland) is not very frequently captured. It is not plentiful anywhere, is rather shy and extremely rapid in its movements. Even large specimens yield only a small quantity of venom on milking, so that it is difficult to obtain it in any considerable amounts.

Tidswell[1] in 13 observations obtained yields of dry venom ranging from 4 to 5·5 milligrammes (average 4·8 milligrammes); N. H. Fairley and Splatt[2] obtained no venom at all from seven snakes two of which were of large size; Kellaway[3] in 62 milkings from 13 snakes obtained a maximal yield of 7·2 milligrammes and an average yield of 2·1 milligrammes of dry venom. A later series of 35 milkings at this Institute by Thomson also gave an average yield of about 2 milligrammes and a maximal yield of 9·9 milligrammes.

Very little appears to be known concerning the toxicity of this venom. Tidswell[1] found that the lethal dose by subcutaneous injection in rabbits (probably wild) was 0·2 milligramme per kilogram. My own earlier observations[3] showed that the venom contains a very potent thrombin. The certainly lethal dose by intravenous injection in sheep was 0·001 milligramme per kilogram. Subcutaneous or intradermal injection caused death by intravascular coagulation in two of three sheep injected with 0·02 milligramme per kilogram and in three others which received somewhat larger doses.

In the present communication I have recorded a single observation of the effects of the venom in the horse, a few upon monkeys and cats and more numerous ones upon rabbits, guinea-pigs, rats and mice.

The sample of venom used was a pooled one obtained by milking a number of snakes between May, 1929, and January, 1931. This sample to which freshly dried venom was added from time to time as collected was kept throughout in a loosely stoppered vessel over sulphuric acid in a desiccator.

Effects in the Horse.

No attempt was made to ascertain the certainly lethal dose for this species.

A single animal weighing 432 kilograms received subcutaneously a dose of 8·6 milligrammes (0·02 milligramme per kilogram). Within three hours the animal exhibited signs of lassitude and there was some bleeding from the nostrils. After three and one-half hours it became disinclined to stand and within six hours it was paralysed. There was dyspnœa and the breathing was diaphragmatic in type. Death took place nine hours and twenty minutes after the injection.

Post mortem, the blood was fluid in all the vessels. There were numerous petechial hæmorrhages in the heart muscle (particularly evident under the endocardium and in the pericardial fat). There was intense hæmorrhagic oedema of both lungs with many subpleural ecchymoses. There was no sign of any bolus in the œsophagus indicative of bulbar paralysis. The kidneys showed intense hæmorrhage in the medullary zone and there were numerous petechial hæmorrhages in the bladder mucosa, though neither hæmoglobinuria nor hæmaturia was observed.

Locally there was slight œdema round the site of injection, but none of the veins in the vicinity was found clotted, as so often followed subcutaneous injection of this venom in sheep.

The dose of venom used was probably considerably in excess of the certainly lethal dose.

Effects in the Monkey.

The results of the subcutaneous injection of the venom in a concentration of one milligramme per cubic centimetre in a few monkeys (*Macacus rhesus*)

[1]Carried out under a grant from the Commonwealth Government Department of Health.

are set out in Table I. The injections were made into the lower limb just below the knee.

TABLE I.
Showing Results of Subcutaneous Injection in the Monkey.

Weight in kilograms.	Doses in milligrammes per kilogram.	Result.
2·83	0·2	Died in less than 20 hours.
2·42	0·15	Killed when moribund after 7 hours.
2·42	0·1	Survived without symptoms.

These results suggest that the certainly lethal dose for *Macacus rhesus* is of the order of 0·2 milligramme per kilogram.

The symptoms were striking, but not particularly characteristic for this venom.

In the animal which received 0·2 milligramme, ptosis appeared within two hours. Within three hours the hind limbs were paralysed and there was definite weakness in the fore limbs, The animal sat in a hunched position taking no notice of its surroundings. After six hours it was paralysed, lying flat out on its side. The breathing was diaphragmatic. The limbs were in the flexed position and somewhat rigid, pulse 160. respirations 39, pupils normal. After seven hours there was profound collapse, the extremities were cold, though the heart was beating vigorously—132 per minute. The animal was cyanosed and the conjunctival reflex was absent. After ten hours the condition remained the same, except that the pulse was feebler and the pupils were contracted. The animal was found dead the following morning.

The second animal presented a similar sequence of symptoms, commencing with ptosis within half an hour of the injection. There was paralysis of all the skeletal musculature except the diaphragm after four hours and the animal was collapsed and cyanosed with cold extremities. After seven hours an anæsthetic was administered and the phrenic nerves were strongly stimulated with a faradic current without any contraction of the diaphragm occurring, though the muscles innervated by the sciatic nerves still responded to a much weaker stimulus.

In both these animals the blood *post mortem* was fluid and the coagulation time in the latter was seven minutes. There was no obvious hæmolysis and no hæmoglobinuria. The lungs were congested and there were a few small hœmorrhages in their substance, otherwise no gross morbid changes were observed.

Effects in the Cat.

The results of the subcutaneous injection of this venom in a concentration of 1 milligramme per cubic centimetre into a few cats are set out in Table II.

TABLE II.
Showing Results of Subcutaneous Injection in Cats.

Weight in kilograms.	Dose in milligrammes per kilogram.	Result.
2·14	0·5	Died in one hour 20 minutes.
3·68	0·2	Died on the second day.
3·36	0·15	Died in 25½ hours.
2·9	0·15	Paralysed in 24 hours. Died on the second day.
2·13	0·12	Paralysed in 24 hours. Died on the sixth day.
2·27	0·1	Paralysed in hind limbs in 20 hours. Died on the second day.
2·78	0·08	Survived without symptoms.
3·4	0·08	Survived without symptoms.
2·05	0·08	Paralysed in 20 hours. Died on the second day.
2·3	0·05	Weakness and ataxia on the second day, recovered by the third day.

The certainly lethal dose is about 0·1 milligramme per kilogram and the toxicity of the venom for the cat is of the same order as that of the venom of the tiger snake.

The symptoms in those animals which died on the second day were very uniform.

Within twenty-four hours there was flaccid paralysis of all the skeletal musculature except the diaphragm. The mouth and nostrils were wet and the respirations were diaphragmatic. The pupils were normal or widely dilated. The flaccid paralysis persisted and the breathing became feebler and slower, the animals finally dying from failure of respiration. The cat which died rapidly following a large dose, was comatose within a hour with a wet snout, widely dilated pupils and absent conjunctival reflex. There was flaccid paralysis of the muscles which was not quite complete, occasional spasmodic convulsions occurring from time to time. The heart was still beating strongly after death which took place from respiratory failure. The blood was fluid in all the vessels and a soft clot formed *in vitro* in nine minutes. The diaphragm contracted feebly when the phrenic nerves were stimulated with a faradic current, the coils being separated nine centimetres, whereas the muscles innervated by the sciatics contracted strongly with the coils separated twenty-two centimetres.

Post mortem, none of these animals exhibited any very striking features except congestion and occasional hæmorrhages in the lungs. In no case was there any hæmaturia or hæmoglobinuria.

Effects in the Rabbit.

Intravenous injection into a marginal ear vein was made in a number of domestic rabbits. The results are set out in Table III. The largest dose was given in a concentration of 0·1 milligramme and the others in a concentration of 0·01 milligramme per cubic centimetre.

TABLE III.
Showing Results of Intravenous Injection in Rabbits.

Number of animals.	Average weight in kilograms.	Dose in milligrammes per kilogram.	Result.
4	1·29	0·01	All died in between two and three minutes.
8	1·24	0·0025	Five died in three and one-half minutes, one in four, one in seven and one in nine and one-quarter minutes.
7	1·27	0·002	One died in four minutes, one in six, one in seven and one-quarter, one in 12, one in 17½ minutes, one in three and one-half hours and one survived.
1	1·42	0·001	Survived without symptoms.

The certainly lethal dose by intravenous injection is 0·0025 milligramme per kilogram.

The symptoms came on within a minute and were typical of intravascular coagulation which tended to be more extensive after the larger doses. Some of the animals which received 0·002 milligramme per kilogram, had whipped out fibrin in the right heart, but no obvious clotting in the pulmonary veins, portal vein or inferior *vena cava*. Thrombosis in the inferior *vena cava* was only found among the animals which died rapidly after the injection of 0·01 or 0·0025 milligramme of venom.

Results of Subcutaneous Injection in Wild Rabbits.

Since Tidswell's[1] estimation of the certainly lethal dose of this venom injected subcutaneously into rabbits (presumably wild animals, though this is not specifically mentioned) was dependent only upon the results in a few animals, I have made some further observations using the venom injected subcutaneously in the abdominal wall in a concentration of one milligramme per cubic centimetre. The results are set out in Table IV.

TABLE IV.

Showing Results of Subcutaneous Injections into Wild Rabbits.

Number of animals.	Average weight in kilograms.	Dose in milligrammes per kilogram.	Result.
2	0·77	0·3	Both died in about two hours.
8	1·1	0·25	One died in one and one-quarter hours, one in one hour 20 minutes, one in one and one-half hours, one in one hour 40 minutes, one in one hour 50 minutes, one in one hour 55 minutes and two in a little over two hours.
10	1·13	0·2	One died in one hour and six minutes, one in one hour eight minutes, two in one hour 20 minutes, one in one and one-half hours, one in one and three-quarter hours, one in two hours, one in two and one-half hours and two survived.
8	1·12	0·15	Two died on the first day, three on the second day and three survived.

The certainly lethal dose is a little more than 0·2 milligramme per kilogram.

The symptoms in those animals which died in one to two hours were of gradual onset. After about an hour the rabbits became collapsed and progressively weaker with flaccid paralysis of the muscles, which was not complete, since in some animals there were feeble convulsive movements from time to time for ten to twenty minutes before the respirations finally failed. Others died quietly, the head falling over tonelessly, the corneal reflex being lost and the pupils widely dilated some time before death.

Post mortem, the heart had invariably ceased to beat when the respirations finally failed and a small quantity of whipped out fibrin was always present in the right heart. The lungs were congested, but in none of the animals were thrombi demonstrated in the pulmonary vessels. The portal vein and the inferior *vena cava* always contained fluid blood and the clotting time was normal—three to five minutes. No thrombosis was found locally at the site of injection, though as in the sheep the diffusible thrombin had evidently escaped into the circulation.

It is unlikely that the observed paralysis and convulsions were due to the cardiac *ante mortem* clot with resultant emboli in the pulmonary and cerebral vessels, since the symptoms were too uniform. They were probably due to the neurotoxic action of the venom.

When death resulted in such short periods as one to two hours, there was no evidence of any peripheral action of the venom. The diaphragm contracted actively when the phrenic nerves were stimulated with a faradic current immediately after death (distance between the coils of 35 to 45 centimetres) and were no less sensitive to such stimuli than the sciatic nerves.

When the venom causes death in so short a time, this result cannot be due to failure of respiration from peripheral action of the venom.

Effects in the Guinea-Pig.

The results of the subcutaneous injection of the venom in the guinea-pig in a concentration of 0·01 milligramme per cubic centimetre are set out in Table V.

The certainly lethal dose for guinea-pigs of this weight is 0·0025 milligramme per 100 grammes or 0·025 milligramme per kilogram. For this species the venom is slightly less toxic than that of the tiger snake. It is powerfully neurotoxic and the symptoms produced are closely similar to those caused by injections of this latter venom.

TABLE V.

Showing Results of Subcutaneous Injection in Guinea-pigs.

Number of animals.	Average weight in grammes.	Dose in milligrammes per 100 grammes.	Result.
16	238	0·0025	Four died on the first and 12 on the second day.
14	225	0·002	One died on the first, six on the second, four on the third day and three survived.

The animals became paralysed before the end of the first day. Their snouts were wet and their coats rough. The paralysis was progressive; "starting movements" were frequent in the later stages and death occurred from respiratory failure. Neither hæmaturia nor hæmoglobinuria was observed in any of them and the only obvious *post mortem* findings were congestion and hæmorrhages in the lungs with occasional patches of collapse. In a few animals there were hæmorrhages into the bowel. Excess of bile-stained fluid in the small intestine was frequently noted.

Effects in the Rat.

The rats used were of the same breed as those used in testing the other Australian venoms. They were white, black and parti-coloured. The results of both subcutaneous and intravenous injections were studied in this species (Table VI). For the former the venom was injected into the flank in a concentration of 0·2 milligramme per cubic centimetre.

TABLE VI.

Showing Results of Subcutaneous Injection in Rats.

Number of animals.	Average weight in grammes.	Dose in milligrammes per 100 grammes.	Result.
17	134	0·08	Thirteen died in less than 18½ hours, one in 20 hours, two in 22 hours, and one in 24 hours.
12	109	0·06	Ten died in less than 20 hours, one in 26½ hours, and one later on in the second day.
12	117	0·04	Seven died in less than 20 hours, one in 26½ hours, two later on in the second day and two survived.
10	131	0·02	Three died during the first day and the remainder survived, some after showing severe symptoms.

The certainly lethal dose is about 0·06 milligramme per 100 grammes or 0·6 milligramme per kilogram.

The symptoms were predominantly neurotoxic, though dyspnœa and wetness of the snout were common features. Death took place from respiratory failure. Neither hæmoglobinuria nor hæmaturia was observed in any of these animals.

Since the rat is very sensitive to haemotoxic venoms, the absence of any such effect with this venom suggests that it has but little hæmotoxic activity.

The Results of Intravenous Injection in Rats.

The venom in a concentration of 0·02 milligramme per cubic centimetre was injected into a lateral caudal vein of rats. The results are set out in Table VII.

The certainly lethal dose is rather more than 0·006 milligramme per 100 grammes or 0·06 milligramme per kilogram and the subcutaneous intravenous index for the rat is less than 10.

TABLE VII.

Showing Results of Intravenous Injection in Rats

Number of animals.	Average weight in grammes.	Dose in milligrammes per 100 grammes.	Result.
2	170	0·015	Both died in about one minute.
9	114	0·01	One died in nine minutes, one in 12, one in 16 and two in 20 minutes, one in an hour and three in less than 16 hours.
10	114	0·006	One died in six minutes, one in 15 minutes, one in 16 minutes, one in 20 minutes, one in 29 minutes, four in less than 16 hours and one survived.
13	115	0·005	One died in six minutes, one in 10 minutes, two in 15 minutes, one in 19 minutes, one in 29 minutes, one in 40 minutes, two in less than 16 hours, one in 18¼ hours, and three survived.
12	126	0·004	One died in six minutes, one in eight minutes, one in one hour, one in one and one-half hours and eight survived.

All these animals within a minute after the injection had symptoms typical of intravascular thrombosis and in all those which died rapidly thrombi were found in the heart, lungs or portal vein. In the half dozen which were found dead next morning, it was difficult to be certain that death had resulted from intravascular thrombosis and none of them had congestion or hæmorrhage in the small intestine or splenic enlargement suggestive of portal thrombosis.

Effects in the Mouse.

The mice used in these experiments were of mixed breed some black, fawn, grey and albino and some parti-coloured. They were the same strains as those used in testing the other Australian snake venoms. The venom was injected in a concentration of 0·01 per cubic centimetre. The results are set out in Table VIII.

TABLE VIII.

Showing Results of Subcutaneous Injection in Mice.

Number of animals.	Average weight in grammes.	Dose in milligrammes per 20 grammes.	Result.
5	21	0·008	All died in less than 18 hours.
10	20	0·005	One died in three and one-half hours, eight in less than 18 hours and one one the second day.
18	19	0·004	16 died in less than 20 hours and two survived.
20	20	0·003	Three died in less than 18 hours, one in 20 hours, seven in less than 21 hours, two in 22 hours, one in 25 hours, two on the second day, one on the third day, one on the sixth day, one on the eighth day and one survived.
26	19	0·002	Eight died on the first day, one on the third day and 17 survived, some after exhibiting severe symptoms.

Though the number of animals injected is insufficient to determine it with accuracy, the certainly lethal dose is about 0·005 milligramme per 20 grammes of body weight, or 0·25 milligramme per kilogram.

The mice which died exhibited symptoms which appeared to be neurotoxic, though it was difficult in these small animals to exclude the possibility of intravascular thrombosis. They became paralysed and died from failure of respiration. As in rats, hæmoglobinuria did not occur following the injection of this venom. *Post mortem*, the only striking lesions were hæmorrhages in the lungs. There were no obvious changes at the site of injection.

Results of Intravenous Injection in Mice.

A few mice of the same breed were injected intravenously with venom in concentrations of 0·002 milligramme per cubic centimetre for the two larger doses and of 0·001 milligramme for the smaller one. The results are set out in Table IX.

TABLE IX.

Showing Results of Intravenous Injections in Mice.

Number of animals.	Average weight in grammes.	Dose in milligrammes per 20 grammes.	Result.
4	21	0·0003	One died in one and one-half minutes, one in two minutes and two in four minutes.
14	21	0·0002	Two died in two minutes, three in three minutes, one in three and one-half minutes, four in four minutes, one in six minutes, one on the second day and two survived.
6	18	0·0001	All survived without any symptoms.

The certainly lethal dose is rather more than 0·0002 per 20 grammes or 0·01 per kilogram. The subcutaneous intravenous index is about 20 for this species.

All the mice injected with the two larger doses developed the classical symptoms of intravascular coagulation within a minute or so after the injection.

The general results of the injection of this venom in various species are summarized in Table X.

TABLE X.

Certainly Lethal Doses of Brown Snake Venom in Various Species.

Animal.	Certainly lethal dose in milligrammes per kilogram.		Approximate subcutaneous intravenous index.
	By subcutaneous injection.	By intravenous injection.	
Sheep	0·02	about 0·001	abou 20
Monkey	about 0·2	—	—
Cat	about 0·1	—	—
Rabbit	more than 0·2	0·0025	—
Guinea-pig	0·025	—	—
Rat	0·6	more than 0·06	about 10
Mouse	0·25	more than 0·01	about 20

The venom is powerfully neurotoxic for all these species, the sheep and guinea-pig being particularly susceptible. In this respect it is of the same order of toxicity as the venom of the tiger snake, but is somewhat less potent. The thrombin of brown snake venom is very active and is on the whole a little more potent than that of the tiger snake.

The thrombin is also potent *in vitro*. Miss Williams has carried out some tests for me in which 0·2 cubic centimetre of citrated plasma was added to varying doses of venom in 0·1 cubic centimetre of saline solution. In Table XI are set out the coagulation times in minutes of the plasma of various species.

TABLE XI.
Coagulation Time of Various Plasma by Brown Snake Venom in Vitro.

Dose of venom in milligrammes.	Human.	Horse.	Sheep.	Rabbit.	Guinea-pig.
0·1	1	2	2	¾	1
0·01	1½	2	2	¾	1½
0·001	60 (soft)	3	4	1	—
0·0001	—	15	16	4	—
0·00001	—	70	—	30	—
0·000001	—	—	—	56	—
0·0000001	—	—	—	—	—

The thrombin of this venom is very unstable in acid solution. When the venom in a concentration of 0·2 milligramme per cubic centimetre is dissolved in saline solution at pH 3·8 it rapidly loses its activity. A dose of 0·02 milligramme per kilogram, injected intravenously into domestic rabbits, does not regularly kill them and one of 0·1 milligramme per kilogram causes no mortality injected intravenously in rats, the certainly lethal dose of venom given under these conditions being rather more than 0·15 milligramme per kilogram. The solution tested in rabbits had lost more than 90% and that used in the rat experiments had lost at least 60% of its coagulant activity.

Discussion.

The observations on animals show that the venom has only a slight hæmolytic activity. This is confirmed by the results of *in vitro* tests carried out upon 3% suspensions of the washed red blood corpuscles of various species, and also upon suitably diluted defibrinated blood. With the use of a system comprised of 0·1 cubic centimetre of venom solution, 0·1 of red blood cell suspension and 0·1 cubic centimetre of saline solution, no hæmolysis was observed with doses of 1·0 milligramme, 0·1 milligramme, 0·05 milligramme and diminishing doses to 0·006 milligramme of venom with human red blood corpuscles nor with those of the sheep and rabbit. Washed horse red blood cells were not hæmolysed even by one milligramme of venom, but defibrinated blood or blood to which horse serum had been added was partially hæmolysed after two and one-half hours with 1·0 milligramme and 0·5 milligramme of venom. The washed red blood cells of the guinea-pig were completely hæmolysed in two and one-half hours by one milligramme and after twenty hours hæmolysis had occurred with 0·1, 0·05, 0·025 and 0·0125 milligramme of venom.

These observations on the toxicity of brown snake venom throw light on the deaths which occur in man following bites by this snake. Summarizing the figures obtained by Tidswell and Ferguson, Hamilton Fairley[4] has found that the mortality rate for bites by this species in man is 8·6%. In view of the very poor venom apparatus possessed by the brown snake and the small venom yields in captivity,[1] the venom would need to be highly potent to cause any mortality among large animals.

It is of course possible that the venom yields obtained in captivity are misleading and that in a free state the snake injects more venom than we are usually able to collect by milking. This is rendered more likely by the fact that these snakes rarely bite willingly through the rubber dam on the capsule and that "milking" is unsatisfactory unless the snake is itself attempting to express venom.

[1] We have recently obtained a primary venom yield of 67·2 milligrammes of dry venom from a snake nearly 7 feet in length.

Conclusions.

1. The venom of the brown snake (*Demansia textilis*) is highly potent though somewhat inferior in this respect to the venom of the tiger snake.
2. It has little hæmolytic activity but is powerfully neurotoxic in action. Observations on the monkey and cat suggest that this last effect is partly peripheral.
3. The thrombin of the venom is very powerful both *in vivo* and *in vitro*. When injected subcutaneously or even intradermally, it readily enters the vessels and causes death in some species by intravascular coagulation. It is extremely unstable in acid solutions.
4. The high toxicity of the venom accounts for the mortality caused by bites in man which is unexpected in view of the poor venom yields and biting apparatus of this snake.

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(2) N. Hamilton Fairley and Beryl Splatt: "Venom Yields in Australian Poisonous Snakes," THE MEDICAL JOURNAL OF AUSTRALIA, March 16, 1929, page 347.
(3) C. H. Kellaway: "Local Venesection in the Treatment of Snake-bite: An Experimental Study," THE MEDICAL JOURNAL OF AUSTRALIA, September 13, 1930, page 356.
(4) N. Hamilton Fairley: "The Present Position of Snake Bite and the Snake Bitten in Australia," THE MEDICAL JOURNAL OF AUSTRALIA, March 9, 1929, page 310.

Reviews.

THE USE OF HIGH FREQUENCY CURRENTS.

IN "High Frequency Practice" Dr. Burton Baker Grover sets out to tell the complete tale about diathermy in its relations to medical practice generally.[1] He does this with considerable success, as the publication of this, the sixth, edition testifies. It is a pity that the author gives his book such a loose title. "High frequency practice" might refer to meals, movements of the bowels or even to alcoholic refreshment. The variations from the last edition are neither important nor numerous. A larger glossary of physical therapy terms has been added, the value of which scarcely seems to justify the expenditure of forty-two pages. A further forty pages are also taken up with that American abomination known as a "quiz" and consisting of question and answer based on the preceding text. This is almost entirely repetition and savours too much of the kindergarten to be included in a scientific work.

The book begins with a chapter on "The New Physics," which traverses in jolting fashion the electron theory, radio-activity and some aspects of radiation. We learn on page twenty-six: "that particles are deflected from the anode of an x-ray tube and that their penetrating qualities are due to their extreme speed. It is reasonable to assume that the sun's rays are composed of an actual substance. The extreme speed of 186,000 miles per second gives to light its penetrating character." This is certainly new physics.

On the physics of high frequency currents and on diathermy generally, the author is far more convincing

[1] "High Frequency Practice with Appendix for Practitioners and Students," by B. B. Grover, M.D.; Sixth Edition; 1931. Kansas City: The Electron Press. Royal 8vo., pp. 648, with illustrations. Price: $7.50 net.

and apparently more at home with his facts. Most of the latest work is mentioned, but some without the discussion it deserves. Autocondensation is dealt with completely, though the statement that very high frequencies sedate the nervous system whilst lower frequencies act on metabolism could well be elaborated and evidence brought forth to support it. It is gratfying to learn that Sir Clifford Allbutt believed autocondensation "the most valuable immediate aid we possess for hyperpiesia."

High blood pressure and cardiovsacular diseases are treated at some length, and on the whole satisfactorily, though there is much that is contentious included.

Concerning diseases of the respiratory system, the application of diathermy to patients with pulmonary tuberculosis is recommended, with due caution as to dosage. Theoretically, the increased circulation should be beneficial and on this ground the writer puts up a good case for the method. Diathermy in pneumonia, of course, receives ample mention.

Taken as a whole, this book is a fair and reasoned exposition of the use of high frequency currents in medicine, though in parts discursive and disfigured by dogmatic assertions, the authority for which is not evident either in this work or in other literature. On page 449, for example, we may quote the interesting but unadorned fact that: "Albuminuria due to nervous disturbances is benefited by the monoterminal application of high frequency current from a non-vacuum electrode to the fourth and fifth dorsal vertebræ." Our mental reaction to this is that it is good as far as it goes, but that it does not go far enough In short, this book is like the curate's egg—"good in parts." The book is well worth reading by those who are interested in the subject, but it can be recommended only to those of mature age and sound judgement who can read with discretion.

CRIPPLES.

IN "Crippled Children" E. D. McBride has produced a book that will prove useful to those working on the problem of the crippled child.[1] It is primarily written to supply "those interested in the care and treatment of crippled children with such knowledge and information as the orthopædic surgeon would deem advantageous to have presented to them. This information is for the nurse in her training and practice and for social workers." Clear descriptions of orthopædic conditions are given, with their causes, symptoms and treatment. There are detailed instructions for the pre-operative and post-operative care and the use, fitting and care of the various splints and plasters. The teaching in general follows the best conservative American schools. It is therefore more remarkable that in the chapter on infantile paralysis the importance of prolonged recumbency with careful muscle exercises is not emphasized, but that early ambulatory splints are advocated. Apart from this, however, the teaching is sound. A short appendix with definitions of common orthopædic terms gives a handy reference for lay readers. The book can be recommended to all those interested in the nursing and after-care of the crippled child.

TREATMENT BY RADIATION.

DISASTER lies in the way of those who attempt the practice of radiation therapy without the necessary knowledge and training. In "Practical Radiation Therapy" Dr. Ira I. Kaplan, Radiation Therapist at the Bellevue Hospital, defines the principles of radiation and the methods of carrying it out as it is practised at that hospital.[2] The writer deals with his subject in a clear and concise manner.

Radiation is no longer a haphazard, go-as-you-please method of treatment. Certain rules and laws govern it, although there is scope for variation within defined limits. Owing to the dangers which lie on either side of the legitimate path, it is essential that anyone making use of radiation should be able to follow that path. This means that an apprenticeship is essential and study a *sine qua non*. Such a work as the one under review is of great value both to the intending radiologist and to those who are already practising this branch of medicine.

In the chapter on practical radiation Dr. Kaplan details cases that have been treated with the X rays and with radium at the Bellevue Hospital, and gives the methods employed and the dosages. The conditions treated cover a wide extent, demonstrating the usefulness of radiation, whether as an adjuvant to the surgeon's knife or as an independent entity.

A special chapter on applied X ray physics, by Carl B. Braestrup, provides the fundamental knowledge of the sources and action of radiation, which is necessary to the understanding and application of this form of therapy.

Numerous illustrations show in a practical manner the results of treatment by radiation, while a short chapter deals with endothermy.

Notes on Books, Current Journals and New Appliances.

SEX RESEARCH.

THE Second International Congress for Sex Research was held at London in August, 1930. Professor F. A. E. Crew was President and Dr. B. P. Wiesner Secretary-General. The proceedings have been published.[1] The papers, which are in English, German, French and Italian, are divided into five groups. The first group has to do with biology—general and mammal, bird, amphibian, insect, botany. The second part is concerned with general, male, female and pituitary hormones. The third part is devoted to therapy, the fourth to contraception and the fifth to sociology. The arrangement is admitted in the preface to be somewhat arbitrary. It is obviously impossible to make any attempt to review the several papers. The volume cannot fail to interest those who study this subject.

THE VETERINARY JOURNAL.

ONE of the subjects to which it is said that veterinarians do not pay sufficient attention, is physiology. *The Veterinary Journal* for October, 1931, is styled a "Special Physiology Number." It contains articles on the diagnosis of equine pregnancy by biological test, the coordination of the reproductive processes, the regulation of the acid-base balance of the body, stores of blood, and the reticulo-endothelial system. Medical graduates who are interested in comparative medicine, will find this issue of *The Veterinary Journal* useful.

THE AUSTRALIAN MUSEUM MAGAZINE.

The Australian Museum Magazine for October-December, 1931, has been received. Medical practitioners who do not know this quarterly publication, are advised to make its acquaintance. This number contains articles on Australian wild life, attack and defence among insects, ground-dwelling birds, Queen Nefertiti, "Furred Natives of our Coastal Regions," skates and rays, and aboriginal flaked implements. This magazine is intended for the general public.

[1] "Crippled Children, Their Treatment and Orthopedic Nursing," by E. D. McBride, B.S., M.D., F.A.C.S.; 1931. Saint Louis: The C. V. Mosby Company. Royal 8vo., pp. 280, with 159 illustrations. Price: $3.50 net.

[2] "Practical Radiation Therapy," by I. I. Kaplan, B.S., M.D., with a special chapter on Applied X Ray Physics, by C. B. Braestrup, B.Sc., P.E.; 1931. Philadelphia: W. B. Saunders Company; Melbourne: James Little. Royal 8vo., pp. 354, with illustrations. Price: 36s. net.

[1] "Proceedings of the Second International Congress for Sex Research," London, 1930; Edited by A. W. Greenwood; 1931. Edinburgh: Oliver and Boyd. Royal 8vo., pp. 648, with illustrations. Price: 21s. net.

The Medical Journal of Australia

SATURDAY, DECEMBER 12, 1931.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

THE BENDIEN CANCER TEST.

A FEW months ago news reached Australia by cable that a European scientist, Dr. S. G. T. Bendien, had gone to London to demonstrate his method of recognizing cancer. Shortly afterwards medical journals from the Old Country confirmed the cabled news, and it was announced that Dr. Alfred Piney, Secretary of the Investigation Committee of the British Cancer Campaign, had begun to investigate the method. Work of this kind, heralded in such a way, was bound to attract attention. Although some people, allowing their judgement to be warped by their own wishes or by these clamant announcements, were prepared to believe that the so far unattainable had been attained, the majority decided to suspend judgement and to await confirmation. They were the more determined in this course by seeing in the pages of a well-known London illustrated weekly pictures of Dr. Bendien in various attitudes, at work and at leisure, and surrounded by inquiring scientists. Caution in accepting the results of research, and cancer research above all else, is wise, and the present instance offers no exception. In this week's issue we publish the results of attempts to confirm Bendien's work by Miss Freeman and others at the Walter and Eliza Hall Institute for Research in Pathology and Medicine. By a coincidence the latest English mail brings a book by Dr. A. A. Miller, entitled: "Bendien's Diagnostic Methods for Cancer and Principles of Treatment."[1] Dr. Bendien has written a book, *"Spezifische Veränderungen des Blutserums."* Medical practitioners who are not at home in the German language, may look on Dr. Miller's work at authoritative, for Dr. Bendien has written a foreword and given it his *imprimatur*.

Dr. Miller points out that in Dr. Bendien's opinion two factors are essential for the development of cancer: (i) an extrinsic factor, a local persistent irritation; (ii) an intrinsic factor, a higher negative electric charge than normal. Dr. Bendien claims to have demonstrated the intrinsic factor with his modified cataphoresis apparatus. This consists of a U-tube filled with colloidal serum protein and water. Under the influence of an electric field the surface of separation between the serum protein and water begins to move. The displacement is measured by a millimetre scale and its amount is supposed to give an indication of the electric charge of the serum. "Assuming that the cancerous process causes an alteration in the colloidal charge of the tissues immediately concerned, one might imagine that a potential difference is thus created between it and the serum, and that they may be considered tuned one to the other." Dr. Miller states that Dr. Bendien has examined a great number of specimens of serum, and that during these investigations, which consisted principally in flocculating the serum with a special solution, he found that the same pathological condition always produced a more or less typical flocculation. In this way he discovered what he regards as a characteristic precipitation in cancer serum. As far as the test itself is concerned, readers are asked to study the paper by the workers from the Walter and Eliza Hall Institute. Their conclusion that "the precipitation of serum proteins in the Bendien test is not sufficiently specific to be of decisive value in the diag-

[1] "Bendien's Diagnostic Methods for Cancer and Principles of Treatment," by A. A. Miller, M.D., with an introduction by S. G. T. Bendien; 1931. London: H. K. Lewis. Crown 8vo., pp. 79, with one table and nine illustrations. Price: 3s. 6d. net.

nosis of cancerous conditions" should be read in conjunction with the conclusions of Smith, Holiday and Marrack in *The Lancet* of August 29, 1931, that "the spectrophotometric method applied to Dr. Bendien's solutions has no value in the diagnosis of cancer."

Not only has Dr. Bendien elaborated a test for malignant disease, but he has also obtained a serum which he uses for treatment. Before this is discussed it must be explained that he describes a protein in the serum, which he calls "normallabilin." According to Dr. Bendien, the serum globulin is changed by the cancerous process. The precipitation in the flocculation test is not due to the presence of a new substance, but to the specific alteration of the globulin. This precipitate is described by Dr. Miller as consisting of a special protein which is formed as the result of cancer metabolism (this is thermostable) and of a protein which is dissolved when heated to 56° C. for half an hour. This second protein is "normallabilin." Dr. Bendien bases his principles of treatment on two observations. The first is that cancer patients with a high "normallabilin" content of the blood are "clinically less malignant" than patients with a low one. His treatment aims at increasing the amount of "normallabilin" in the blood. The second principle is that the electrical (colloidal) charge of cancer serum is different from normal; the treatment consists in altering this charge so as to balance it. Dr. Miller states that in carrying out his treatment Dr. Bendien makes certain preliminary investigations: (i) He takes the patient's blood. (ii) He performs the flocculation test and identifies the presence of "normallabilin." (iii) He identifies the electric charge of cancer serum. (iv) He identifies the electric charge of the serum to be administered, and prepares it according to preliminary findings. As far as actual treatment is concerned, Dr. Miller goes on to state that if the cancer is "active" and if the tumour is operable, it should be removed. The source of the specific protein is thus eradicated. If the tumour is inoperable and the patient's general condition is good, venesection is performed to decrease the amount of "specific protein" in the blood. If the patient's condition is bad, blood transfusion is performed in order to dilute the "specific protein." In either case: (i) Dr. Bendien administers his therapeutic serum, a hypodermic injection being given every day. (ii) The patient is dieted in order to influence the electric charge of the blood. (iii) Hydrotherapy is administered. (iv) Colonic lavage is given to eliminate toxic products. Unfortunately, as Dr. Miller states in the preface of his book, Dr. Bendien has not published the formula of his serum; "he is aware that it is not yet perfect and that its composition might have to be modified." Dr. Miller makes the gratuitous statement that as a result of Dr. Bendien's treatment the glands first disappear and the tumour diminishes in size and shrinks very gradually. He gives no detailed case reports or other evidence to justify this statement. No exception can be taken to the possibility of the occurrence of changes in the protein constituents of blood in cancer; the Bence Jones proteins found in the urine in some cases of leuchæmia and in certain types of bone tumour indeed suggest the possibility. That Dr. Bendien has discovered a specific protein in the blood of cancer patients cannot be regarded as proven; nor, in view of the work reported from the Walter and Eliza Hall Institute, and in view of the findings of Smith, Holiday and Marrack, can it be held that Dr. Bendien has discovered a test specific for carcinoma. Further, until he tells the scientific world how he prepares his serum he is not likely to gain the confidence of serious workers in any country, nor will his alleged cures arouse their enthusiasm.

Current Comment.

UNEXPLAINED HEART FAILURE.

HEART failure has been defined as "failure of the heart to maintain an efficient circulation." The study of the mechanism of heart failure opens up a very wide subject. In many cases it would seem to involve merely the solution of a number of problems, possibly very intricate, of biophysics, but there are a very large number of occasions when it is felt that some agent not yet hypothecated is responsible. In these columns the story of the Greek courier who ran from Marathon to Athens was recently cited and the cause of his heart failure was considered. The incident might be worth further consideration. He died immediately on

reaching his objective. What was the reason for the dramatic coincidence? Had the distance been a furlong shorter, would he have had a chance of surviving? Had his goal been a furlong further, would he have reached it? Did emotion play any part as a cause of death?

At the Baltimore meeting of the American College of Physicians, March, 1931, H. A. Christian, discussing chronic non-valvular disease of the heart, failed to find any explanation for the obvious fact of death from cardiac failure, or, in many cases, even of the observed symptoms, in patients grouped under this diagnosis.[1] Careful study did not show any ætiological factor in evidence for all of the group, nor did it reveal any pathological lesion which would consistently explain the progressing cardiac failure that led to the patient's death. In the gross the heart muscle looked strikingly well nourished, was normal in appearance and appeared powerful as a muscle. Christian quoted the findings of G. Fitzhugh at the Peter Bent Brigham Hospital in 228 hearts examined at autopsy. Slight or no narrowing of the coronary artery was found in 36·9%, moderate narrowing in 28·3%, considerable narrowing in 35·1%. Microscopical focal fibrosis was found in 68·6%, 22·5% and 8·9% of these.

At the same meeting S. Weiss, in a paper entitled "Circulatory Adjustments in Heart Disease: A Concept of Circulatory Failure," set down conclusions based on recent investigations. He believes that a majority of the symptoms and signs of the patient with cardio-vascular disease depend rather on the state of the circulation than on the heart. His observations indicate, however, that although circulatory failure may be independent of heart disease, as in shock, toxæmia and certain diseases of the nervous system, the local disturbance of the heart is primarily responsible for the circulatory changes and contradict the suggestion that primary changes in the peripheral circulation, such as primary disturbance in lactic acid metabolism, are also responsible for failure of the circulation in heart disease. At the onset of circulatory failure the main symptom referable to the circulation is dyspnœa, and, in his opinion, the mechanism underlying this dyspnœa is the centre of the problem. He found, on examining the peripheral circulation at this stage, that the arterial, capillary and venous blood pressures were unaltered, the cardiac output per minute normal or slightly reduced, and the arterio-venous oxygen differences of the arms and legs and the lactic acid content of the arterial and venous blood normal. Even if dyspnœa was induced by exercise there was no difference between the peripheral circulation of the cardiac patient and the normal individual performing the same exercises. There is, however, an early reduction in the vital capacity of the lungs; simultaneously, or somewhat later, the blood flow through the lungs is retarded and the residual air may be increased, both relatively and absolutely, often at the expense of the reserve air, which decreases. According to Weiss, in progressive failure of the left ventricle the pulmonary arterioles widen and a reserve capillary bed opens in the lungs, which makes it possible to maintain a normal or approximately normal volume of blood flow through the lungs per unit of time without much added burden to the heart, but this opening of new capillaries under normal or higher than normal tension induces a functional emphysema of the alveoli and an early decrease in vital capacity. He offers this theory in explanation of the development of dyspnœa in heart disease, and points out that numerous animal experiments support the contention that dyspnœa in the early stage of circulatory failure is produced through nervous communications between the pulmonary system and the medulla, and not by local chemical changes within the respiratory centre. He contends that even in the later stages of circulatory failure dyspnœa is still a local pulmonary problem, even though substances such as lactic acid increase it by central stimulation. He refers to a group of patients in whom none of the factors of precipitated arrhythmia, coronary occlusion, embolism, uræmia or bronchopneumonia, which cause abrupt death, are found—patients who, although the circulatory functions are still fairly well maintained, exhibit sudden changes in clinical appearance and die within a few hours, some with evidence of vasomotor collapse and shock. He speculates whether "the persisting dyspnœa, cardiac pain, rapid heart and other factors subject the central nervous system to an abnormal bombardment, thus causing death through a failure of the vasomotor and perhaps other nerve centres rather than through extreme congestive circulatory failure."

There appears in this description something which may be applied to the state of the Marathon runner. Most cardiologists and pathologists, however, would maintain that the myocardium of the Marathon runner was not normal, and they would probably be right. The "disordered action of the heart" ("D.A.H.") of the war was sometimes attributed, not to physical overstrain, but to repression of fear by a nervous soldier who was determined not to give way to it, and to the resulting over-activity of the suprarenal glands. There is no evidence to support the view that an outpouring of adrenalin could cause cardiac disturbance resulting in death. Within recent years evidence was given at an inquest on an anæsthetic fatality that the patient had merely inhaled some *eau de Cologne* poured on to the mask as a preliminary deodorant. This is difficult to explain. *Status thymico-lymphaticus* must be excluded in view of the recent report of the Medical Research Council. In many instances an explanation for sudden and apparently inexplicable deaths would be found if meticulous and exhaustive *post mortem* examinations were made. In the theory advanced by Weiss an initial abnormality of the heart muscle must be postulated. His views are, after all, speculative; this must be remembered. What is needed is definite research, which must not be confined to the heart alone.

[1] *Annals of Internal Medicine*, August, 1931.

Abstracts from Current Medical Literature.

RADIOLOGY.

Kummell's Disease.

F. W. O'BRIEN (*Radiology*, October, 1931) gives a brief review of the literature and discusses Kummell's syndrome. This symptom complex was originally described as a rarefying osteitis of a vertebral body following trauma without there necessarily having been any fracture. However, most of Kummell's work was done before it was possible to secure satisfactory lateral views of the vertebræ. In the author's opinion the condition which Kummell has described is in reality unrecognized and hence untreated compression fracture of a vertebral body—unrecognized because of no cord symptoms, unsatisfactory or no X ray examination, and the erroneous belief that if a patient has suffered only a slight trauma and can walk without great discomfort there cannot be a fracture of the spine.

The Post-Pneumonic Lung.

ALAN L. HART (*The American Journal of Roentgenology*, September, 1931) discusses in considerable detail the pathology and the X ray findings of the various types of pneumonia. Chronic lung changes following pneumonia depend on the severity of the acute process, and those pneumonias in which the connective tissue framework of the lung, the walls of the bronchial tree and the vascular and lymphatic channels are seriously damaged or destroyed, exhibit the largest number of chronic pulmonary sequelæ. Among the most important of these are influenzal pneumonia, influenzal pneumonia invaded by streptococci, the bronchopneumonias accompanying measles and pertussis, and Friedländer's pneumonia. In either pneumonia or pleurisy resolution and absorption of the exudate may occur, in which case recovery ensues; and, on the other hand, the pneumonic or pleuritic exudate may not be properly absorbed, in which case organization by fibroblasts and capillaries begins; and, finally, in either pneumonia or pleurisy suppuration instead of resolution may take place. In pleurisy or cases of undrained empyema dense pleural adhesions may obliterate the pleural space and result in impaired mobility of the lung, retraction of the chest, deflection of the heart and mediastinum, and elevation and fixation of the diaphragm. This may lead to slight bronchial dilatation with few symptoms, or to progressive bronchiectasis eventuating in bronchiectatic abscesses. A pulmonic exudate may fail to be properly absorbed and may result in fibrosis at the site of the original inflammatory process and some compensatory emphysema in other parts of the lungs. After liquefaction and evacuation of its contents an abscess may collapse and heal, but if the walls are thick and indurated this cannot occur. Even after apparent clinical recovery small areas of infiltration may persist in the vicinity of an abscess and infection may flare up again later on. Actual lymphangitis may occur especially in pneumonia associated with influenza, measles and pertussis, and this may be the forerunner of interstitial suppuration, which heals with diffuse fibrosis of the lung. As the result of organization of the exudate or suppuration in either lung or pleura following pneumonia, there is a group of terminal conditions that may be expected to develop in a certain percentage of cases. These are: (i) bronchial dilatation and bronchiectasis, (ii) chronic non-tuberculous infections, (iii) abscess cavities, (iv) areas of massive fibrosis resulting from the healing of abscesses, and (v) diffuse fibrosis.

Chronic Appendicitis.

A. G. SCHNACK (*Radiology*, July, 1931) deals with chronic appendicitis. The condition is not limited to the inflammatory type, but the appendix must be a source of irritation capable of giving local and remote signs and symptoms. The term appendico-pathia is suggested as being more appropriate. The author stresses the importance of having all clinical, pathological and X ray evidence in collaboration before a diagnosis is made. The X ray examination is long and requires several visits. The most typical findings may occasionally be erroneous, but this is true of all diagnostic procedures. The diagnosis is in great part one by elimination, and consideration of the lower right quadrant alone is insufficient. The symptoms may be local only, or the chief symptom may be in the upper part of the abdomen or a combination of both may occur. The following conditions must be excluded: hernia or relaxed ring, kidney conditions, muscle strains, spinal arthritis, ovarian and uterine tumours, pelvic inflammatory disease, pregnancy, especially ectopic pregnancy, and diverticulosis. Pulmonary conditions often produce abdominal symptoms. Epigastric distress may be the chief complaint, and this involves the exclusion of gastric and duodenal ulcers as well as gall-bladder disease. The author gives a long description of the clinical symptoms which may occur, and of the pathological changes seen in the organ. Fæcoliths in the appendix may cause transient or persistent symptoms. The question of fixation is often difficult to determine. An examination from various angles is necessary. The author states that the retrocaecal appendix has been over-emphasized as a necessary reason for surgical operation. Fixation, kinking, ball-valve fæcoliths, impacted inspissated fæces, pin worms, all favour appendiceal stagnation and a continuance of a low grade irritated condition in the organ. An appendix already under tension will be tender when the pressure is so applied that its distal end is put under increased tension. Retention of the barium in the appendix for several days after the caecum has cleared has a certain significance, as has also the degree of concentration of the barium in the organ. An appendix containing muco-purulent exudate may receive the barium, but it will show hazy, indistinct outlines. The significance of non-visualization or blocked appendix is considered. A swollen non-visualized appendix may sometimes be palpable and the pain radiate to the opposite side. The indirect reflex Röntgen signs in the stomach, duodenum, large and small bowel are described.

The Relation of the Diaphragm to Gastric Peristalsis.

M. JOANUIDES AND J. LITSCHGI (*Radiology*, October, 1931) conclude that although primarily a muscle of respiration, the diaphragm has a definite extrarespiratory function. It induces an opening and closing of the cardia and it produces a milking contraction on the cardia in the region of the stomach that is automatic and rhythmical. It compresses and lifts up the fundus and in so doing not only changes the form of the stomach, but also influences the intragastric tension. The author assumes that the diaphragm plays an important rôle in the initiation and maintenance of peristalsis in the stomach.

Quantitative X Ray Diagnosis of Pleural Effusions.

A. BOWEN (*Radiology*, September, 1931) concludes that a small pleural effusion may be missed at both physical and X ray examinations, because it may be so situated as to be indiscernible. The fluid must either be in the sulci at the base of the diaphragm or evenly distributed over the surface of the lung in a thin layer. While it is commonly accepted that 400 cubic centimetres may be present without being demonstrable, the author claims that if the X ray examination is made with the patient in the lateral decubitus position, much smaller amounts may be revealed, since, in the absence of adhesions, the fluid will always change its position.

Yeast Infection of the Lungs.

T. R. HEALY AND L. B. MORRISON (*American Journal of Roentgenology*, September, 1931) consider that bronchomycosis is not so rare as is usually supposed and that the reason the diagnosis is not made more frequently is because many of the cases pass for tuberculosis. Infection occurs from inhalation of dust containing the yeast mould, and considering the wide distribution of the fungi, man must possess a high degree of immunity to the infection. In the mild type the condition of the patient is good. There is no fever and the expectoration is usually scanty. This type may clear up spontaneously or, continuing, may turn into the severe type, which closely resembles pulmonary tuber-

culosis. In this severe type the patient becomes emaciated and there is hectic fever and hæmorrhagic expectoration. This type may be fatal. The X ray appearance is similar to that of pulmonary tuberculosis and the diagnosis is based on the absence of the tubercle bacillus and the constant presence of the yeast cell in the sputum. The presence of yeast cells is pathognomonic.

PHYSICAL THERAPY.

Diathermy Treatment of Nephritis.

R. GANTENBERG (*Deutsche Medizinische Wochenschrift*, July 3, 1931) advocates the use of diathermy in the treatment of acute and chronic nephritis. Its value in acute post-scarlatinal nephritis is well known, but it should also be used more widely for all forms of renal inflammation. The duration of each application is fifteen minutes, repeated daily, especially for chronic cases. In normal persons who were given 1,000 cubic centimetres of dilute tea before the diathermy, the author noted a considerable increase in urinary secretion. This also was seen with acute nephritis. In chronic nephritis there may be a diminution in secretion and the author considers that further investigation is required to determine the best dosage in such circumstances to prevent possible renal damage.

X Ray Therapy in Hyperthyreoidism.

FFRANGCON ROBERTS (*The British Journal of Physical Medicine*, July, 1931) supports Falta, who states that all patients with hyperthyreoidism should be treated by X rays unless there is some special contraindication, such as tracheal compression. In Denmark, according to Moeller, radiation is now the favourite form of treatment. Statistics from various quarters show beyond a doubt the beneficial effects of X rays in the great majority of cases. Much of the prejudice against X rays is due to the accidents dating from the days when the method was in its infancy. Now, however, the measurement of dosage has reached such a standard of accuracy that X ray therapy can in competent hands be claimed to be completely free from danger. In this respect it has the advantage over surgical operation, which is not free from risk of fatality and is not invariably successful.

Cancer of the Lung.

N. P. DUANY, L. FARINAS, A. N. SIERRA AND J. A. DEL REGATO (*Vida Nueva*, April 15, 1931) describe nineteen cases of pulmonary cancer. They were all instances of carcinoma. Sarcoma and benign tumours of the lung are rare. Cough, pain, fever, expectoration, loss of appetite, loss of strength, sleeplessness and a sense of thoracic oppression were the symptoms complained of. The diagnosis depends on the coordination of the clinical examination with radiographic investigation. In some cases a biopsy through a bronchoscope can be practised. Treatment was by deep X ray therapy; the prolonged and intensive therapy respected only the skin. At times a dose corresponding to three erythema doses was delivered without accident. The authors are convinced that unless the growth is very advanced, the patient's symptoms improve under X ray therapy—the appetite reappears, body weight increases, and pain disappears. Unfortunately, as a rule, the symptoms recur later and persist. Reference is made to the technique of embedding radium needles or emanation "seeds" into the growth through a bronchoscope, but the authors had not employed this method and admit the difficulty of estimating its value.

Irradiation Treatment of Bone Tumours.

G. E. PFAHLER AND L. D. PARRY (*The American Journal of Roentgenology and Radium Therapy*, June, 1931) classify the therapeutic indications for irradiation treatment of osteogenic sarcoma as follows. Metastatic tumours following a primary tumour in tissues other than bone (breast, thyreoid, prostate, stomach, uterus) call for irradiation. Periosteal fibrosarcoma call for irradiation, probably followed by amputation. Osteogenic tumours, when benign (osteoma, exostoses, chondroma), call for excision; when malignant (periosteal, medullary and subperiosteal, sclerotic, telangiectatic), call for irradiation, possibly followed by amputation. Inflammatory conditions should be irradiated. Benign giant cell tumours (*osteitis fibrosa*, including nasal fibroma, adamantinoma, *myositis ossificans*) call for irradiation. Angiomata, when benign, should be treated by surgical operation; when malignant, by irradiation, probably followed by complete excision or amputation. Ewing's sarcoma calls for irradiation, myeloma for irradiation. The authors agree with Holfelder that the value of histological examination is chiefly for statistical purposes and they have not found it of assured value in determining the sensitivity to irradiation nor in making a prognosis. However, they advise a biopsy in all cases in which the patient will consent, but only after a preliminary irradiation over a period of one month. They have seen no rapid metastasis following biopsy when preliminary irradiation has been given. The response to irradiation in osteogenic sarcoma is remarkably slow. While other tumours show a response after a few weeks, the bone sarcomata may seem to be getting worse for from one to three months and then show improvement. At this stage the authors have generally recommended amputation, but they add that perhaps more patience would have made the amputation unnecessary. From practical observation it seems, therefore, that preliminary irradiation is useful even though operation is decided on at a later date. Basing their opinion upon Holfelder's record of improvement following exacerbation of symptoms after many months, the authors feel less inclined to recommend amputation in the future.

The Treatment of Disfigurements by Physical Methods.

G. B. DOWLING (*The British Journal of Physical Medicine*, June, 1931) describes the treatment of the following skin lesions: (1) Vascular nævi, of which there are three types: (i) the strawberry mark or raised capillary nævus; (ii) cavernous nævus or deep subcutaneous nævus; (iii) flat capillary nævus or port wine stain. The strawberry mark is easily destroyed with carbon dioxide snow. The scar is supple and may be hardly noticeable. The subcutaneous nævi may be treated by electrolysis, electrocoagulation, radium, or by the use of sclerosing injections. At present the treatment of port wine mark is both difficult and unsatisfactory. Three methods are available: (i) Radiotherapy, (ii) freezing, (iii) multiple ignipuncture. In discussing vascular abnormalities other than nævi the author states that stellate angioma and *nævus eraneus* or spider nævus are not nævi, but minute vascular swellings surrounded by a network of arborizing telangiectases. The central swelling may be destroyed easily with a galvano-cautery. Diathermy is equally effective. Senile angiomata are little vascular new growths of about the size of a pin's head, generally situated on the trunk. If desired, they may be easily destroyed with the point cautery or by electrocoagulation. *Adenoma sebaceum* belongs to the group of malformations known as nævi. The condition may be treated by touching each lesion with the blunt point cautery carried to dull red heat or by electrocoagulation. Small moles may be destroyed by electro-coagulation. Moderately large moles may be either excised or destroyed by electrocoagulation under local or general anæsthesia. Warts of psychogenic origin may, according to some authorities, be cured by suggestion. They believe that the successful results of other treatments are due to the suggestion conveyed to the patient. There is no doubt that warts may disappear spontaneously and that they often respond to local treatment with unexpected rapidity. Warts of infective origin may be satisfactorily treated by freezing with carbon dioxide snow or by diathermy. Plantar and palmar warts are best treated by X rays. Some of the horny thickening may be removed as a preliminary step by applying a salicylic acid plaster for a week or ten days. In almost all cases somewhat large doses are necessary. The treatment of periungual warts is by a pastille X ray dose or radium. Seborrhœic warts and senile keratoses may be destroyed by two or three applications of a saturated solution of trichloracetic acid at intervals of a few weeks.

Special Articles on Aids to Diagnosis.

(Contributed by Request.)

IX.

LUMBAR PUNCTURE.

ALTHOUGH the clouds of controversy still overhang the mechanism of formation, circulation and resorption of the cerebro-spinal fluid, much has been learned in this present century regarding the nature of the fluid itself. In health, while maintaining within constant limits the "integrity of a nutritive *milieu*," the cerebro-spinal fluid reacts in the presence of certain diseases of the central nervous system, various intracranial pathological states being reflected in changes in the physico-chemical constituents of the fluid, the estimation of which may aid the establishment of a diagnosis.

It is probable that the part played by the ependymal lining of the chorioid plexus is more complicated and intricate than simply that of a semipermeable membrane maintaining a balance between the passage of crystalloids and colloids in the blood plasma and the cerebro-spinal fluid. But a certain simplicity of osmotic balance may be assumed in studying the qualitative and quantitative reactions of the constituents of the cerebro-spinal fluid in the presence of organic disease of the central nervous system.

Examination of the cerebro-spinal fluid obtained from different loci, both in health and disease, has revealed slight differences in chemical and cytological composition, and it is necessary under certain circumstances to make use of this knowledge and to examine fluid collected both by lumbar and cisternal puncture. But in general the results obtained by examination of fluid withdrawn by ordinary lumbar puncture are of sufficient value for practical diagnostic purposes in the majority of cases.

It should be emphasized here and now that with a few singular exceptions, notably syphilis of the central nervous system, an exact diagnosis cannot be made on the examination of the cerebro-spinal fluid alone. Further, it is undesirable and often grossly misleading to endeavour to build up a diagnosis on the cerebro-spinal fluid findings, however characteristic they may be thought to be. Irritative or inflammatory processes altering the permeability of the hæmato-encephalitic barrier result in changes in the composition of the cerebro-spinal fluid, but the extent and nature of these changes depend on variations of virulence and situation of the lesion. It will be obvious, therefore, that the biochemical estimation of pathological constituents will, in most cases, merely indicate the presence of disease without pointing to specificity. And it is only when regarded ancillary to the clinical symptomatology previously elicited, together with the history volunteered by the patient, that the examination of the fluid obtained by lumbar puncture may assume the importance of an indispensable diagnostic aid.

It is impossible to list the indications for lumbar puncture. Although it finds its chief place as a diagnostic aid in suspected meningitis, poliomyelitis, cerebral hæmorrhage and neurosyphilis, there is scarcely any organic disease of the central nervous system in which lumbar puncture fails to give valuable confirmative evidence; and it should therefore be undertaken almost as a routine in the complete investigation of neuropsychiatric cases. The contraindications are few and are of minor importance in contrast to the value of the information to be obtained. The chief contraindication exists in the suspected presence of cerebral tumours in the posterior cranial fossa, when sudden reduction of pressure has been known to cause death owing to the descent and compression of the medulla in the *foramen magnum*.

Lumbar puncture, unless by particular circumstances rendered absolutely necessary, is contraindicated in patients suffering from erysipelas, measles, scarlet fever or chickenpox. Lumbar puncture is said to be a hazardous procedure in cases of internal hydrocephalus and cerebral œdema; but in my experience a properly conducted lumbar puncture in the recumbent position, with the slow withdrawal of a small quantity of fluid, may be undertaken without trepidation in almost every case.

Complete asepsis and chemical cleanliness are equally necessary in the performance of successful lumbar puncture. A contaminated fluid is useless for diagnostic purposes. It is seldom necessary to remove more than ten cubic centimetres of fluid, as this amount is more than enough for the essentials of examination—a total and differential cell count, total protein estimation and tests for excess globulin, together with a Wassermann test.

Failure to obtain fluid through the lumbar route, in my experience, seldom occurs if one's technique is correct. In most cases of "dry tap" the operator is at fault, the needle having been pushed either too far or not far enough; but occasionally (in purulent meningitis) the density of the exudate clogs the needle and the fluid will not flow. Any lesion which sets up a spinal block above the site of puncture or causes closure of the foramen of Magendi may be responsible for the absence of fluid in the caudal extremity of the spinal canal, the dry puncture itself thereby assuming a diagnostic significance. In certain lesions of the *cauda equina* obliterating the subarachnoid space, and in rare cases of spinal caries, *spondylitis deformans*, the deformity produced may render lumbar puncture at the usual level unsuccessful or even impossible.

The cerebro-spinal fluid in health is a fluid of crystalline clarity, containing a few small lymphocytes and traces of protein. The chemical composition and physical properties vary slightly in different situations and in different individuals within so-called normal limits. In the following tables are set out figures now generally accepted as representing the normal composition of the cerebro-spinal fluid.

Physico-Chemical Constants.

Reaction: pH 7·4 to 7·5 (changes on standing).
Specific gravity: 1006 to 1007.
Freezing point: 0·56° to 0·58° C.
Surface tension: 7·35, 7·15, 7·16 and 7·20 dynes at 20° C.
Refractometric index: 1·33554 at 20° C.
Viscosity: 1·0424 to 1·0489.

Biochemical Constants.

(Estimated in milligrammes per 100 cubic centimetres.)

Protein	20 to 30
Glucose	45 to 85
Chlorides (as sodium chloride)	725 to 750
Urea	20 to 40
Phosphates	1·5 to 2·0
Nitrates	0·5 to 1·0
Calciums	5·7 to 6·8
Magnesium	1·4 to 3·5
Uric acid	0·3 to 1·3

Traces of diastase and lipase and of pituitary hormone.

Physical Changes in the Cerebro-Spinal Fluid in Disease.

It will be obvious that, where a disease process causes irritation of the meninges or alteration of the permeability of the chorioid plexus leading to alteration in the chemistry of the fluid, there will be corresponding changes in such physical characteristics as viscosity, freezing point and refractometric index *et cetera*, and a measurement of these changes, therefore, becomes indicative of pathological change in the biochemistry of the cerebro-spinal fluid.

It is claimed by Levinson that all pathological fluid produces a foam on shaking, which may persist for half an hour or more. The main physical characteristic of some abnormal fluids is the formation of a pellicle on standing. This is a fine fibrin network and indicates the presence of fibrinogen. It is seen preeminently in cases of suppurative and tuberculous meningitis. It is less characteristic of poliomyelitis and has been observed infrequently in certain cases of neurosyphilis. It is always associated with marked protein increase. In very highly albuminous fluids the coagulation may become "massive" and form a complete clot, which, if associated with a yellow coloration, constitutes the syndrome of Froin (*vide infra*).

Colour Changes and Turbidity.

Many pathological fluids are colourless, but a coloured fluid is always pathological. Fluids of a reddish colour are obtained in cases of cerebral or subarachnoid hæmorrhage, cerebro-spinal traumatism and hæmatomyelia. The condition is termed "erythrochromia." The yellowish discoloration, characteristic of the Froin syndrome, is known as "xanthochromia." The phenomenon of xanthochromia may be sometimes observed in cases of cerebral and spinal tumour, spinal caries, cerebral thrombosis and polyneuritis (rarely).

Generally speaking, cerebral hæmorrhage of recent origin causes the cerebro-spinal fluid to appear bright red; when the hæmorrhage is of long standing the colour changes to yellow. It should be remembered that the cerebro-spinal fluid may have become contaminated by accidental pricking of a vein in the operation of lumbar puncture, and it is obviously essential to distinguish a contingency such as this, and the presence of blood originating from a cerebral or subarachnoid hæmorrhage. Although it may be necessary to attempt to obtain a clear fluid by another puncture at a higher level, centrifugalization may serve to show the origin of the blood. In the case of accidental contamination the supernatant fluid is clear, whereas in the case of cerebral or subarachnoid hæmorrhage this fluid is usually tinged yellow.

Changes in colour and turbidity may be due to the abnormal presence of cells or to microorganisms and degeneration products of infective origin. Greenfield has stated as a general rule that "an excess of polymorphonuclear cells over 300 per cubic millimetre gives rise to an obvious turbidity, whereas a lymphocytosis of 500 per cubic millimetre may be present in an absolutely or almost clear fluid."

Turbidity generally indicates acute meningitis—gross in the case of pyogenic or meningococcal meningitis and slight in cases of tuberculous and syphilitic meningitis. It is sometimes seen in cases of cerebral abscess and in suppuration in the cranial air sinuses (Greenfield).

Pressure.

The pressure of the cerebro-spinal fluid, representing a physiological balance between the process of secretion and absorption, is believed to vary with that of the intracranial venous pressure, as demonstrated by the Queckenstedt phenomenon. It is also related to the amount of fluid produced, and will, therefore, following the rule applicable to irritation within a serous cavity, be increased by meningeal irritation.

Slight pressure oscillations may be noted, corresponding to the pulse and respiration rates. These may become exaggerated in highly strung and nervous women or in stout men where there is embarrassment of respiration.

The estimation of cerebro-spinal fluid pressure is of such great diagnostic importance that it should always be carried out by the correct manometric method, as the information gained by observing the rate at which the fluid flows from the needle is likely to be quite fallacious. The patient should be lying horizontally with the head on the same level as the lumbar part of the spine.

Normal pressures vary between 120 and 200 millimetres of cerebro-spinal fluid. Pressures above 200 millimetres indicate definite increase of intracranial pressure, although in some cases pressure of 500 millimetres and even higher may be recorded. Greenfield believes it to be a good rule never to reduce the cerebro-spinal fluid pressure below two-thirds that of the initial pressure, to avoid both the too rapid or the too great lowering of pressure in the suspected presence of a cerebral tumour.

The cerebro-spinal fluid pressure is pathologically increased in cases of cerebral neoplasm, cerebral hæmorrhage, cerebral œdema, hydrocephalus, meningitis of whatever origin, encephalitis, cerebral *lues*, chronic epilepsy and chronic alcoholism. Differentiation between the increased intracranial pressure of a cerebral tumour and hydrocephalus may be achieved by the reduction of pressure following the withdrawal of a small quantity of fluid, when the pressure is seen to fall markedly in the presence of tumour and scarcely at all in the case of hydrocephalus.

Low pressures (that is, 50 millimetres or less) are sometimes encountered, the clinical significance of which it may be impossible to determine, but may be suggestive of a spinal tumour or Pott's disease. Low pressure is recorded typically in those pathological states which cause a "spinal block"—cases in which arachnoidal adhesions, following transverse myelitis or cerebro-spinal meningitis, are tending to obliterate the subarachnoid space above the site of puncture. Should this occur, the information secured from differential pressure readings obtained by combined lumbar and cisternal puncture is invaluable as a diagnostic aid. It is here, too, that the Queckenstedt test finds its chief application. Normally, compression of the jugular veins in the neck causes a marked rise of cerebro-spinal fluid pressure. This will be diminished or absent in cases of spinal block.

Cytological Changes.

The small lymphocyte occurs normally in the cerebro-spinal fluid; the large lymphocyte occurs sometimes; the large mononuclear cell is present occasionally; the polymorphonuclear leucocyte is quite alien to the normal fluid, and its presence connotes some intracranial inflammatory reaction. The presence of the other types of leucocyte, eosinophile cells, plasma cells *et cetera*, appears to the accompaniment of particular pathological states, chiefly neurosyphilis and tuberculous meningitis.

Pleocytosis.—Any number of lymphocytes above three to five per cubic millimetre constitutes a pleocytosis. There are three chief types of cell increase: (i) the mononuclear, consisting of lymphocytes and large hyaline cells only; (ii) the mixed type, consisting of lymphocytes found in combination with polymorphonuclear leucocytes; and (iii) the polymorphonuclear type, consisting almost entirely (75%) of cells of that type.

1. The pure mononuclear lymphocytic reaction is characteristic of neurosyphilis, but may occur in disseminated sclerosis, cerebral abscess, tumour, *encephalitis lethargica* and poliomyelitis. The greater the number of cells present in cases of cerebral syphilis, the more marked is the meningeal involvement, so that, in general, tabes shows the smallest and syphilitic meningitis the largest pleocytosis.
2. The mixed type of pleocytosis is characteristically associated with brain abscess, tuberculous meningitis, poliomyelitis and cerebro-spinal meningitis during recovery. A large pleocytosis of this mixed type is said to occur sometimes in severe syphilitic disease.
3. Moderate leucocytosis of polynuclear type may point to cerebral abscess, either closed or leaking, or some of dural inflammation, but more characteristic is the marked pleocytosis associated invariably with cerebro-spinal meningitis of coccal or bacillary origin.

Chemical Changes in the Cerebro-Spinal Fluid.

Intracranial inflammatory conditions which alter the permeability of the hæmato-encephalitic barrier between the blood serum and the cerebro-spinal fluid, cause characteristic changes in the chemical constituents of the latter, which may assume diagnostic importance. Alteration of the normal meningeal permeability will thus cause a change in the osmotic balance, with a tendency to increasing the concentration of those substances normally present in greater quantity in the blood—phosphates, uric acid, albumin and globulin; to decreasing the concentration of those substances which are normally present in greater quantity in the cerebro-spinal fluid—chlorides. It also permits the passage into the cerebro-spinal fluid of substances normally present only in the blood—fibrinogen, hæmolysin and complement—and the passage of foreign substances injected into the blood stream which normally do not pass through the chorioid plexus (Cohen).

These changes in the cerebro-spinal fluid chemistry give a frequent clue to the particular pathological process responsible for a certain constellation of neurological signs, the clinical significance of which may not otherwise be manifest.

Protein Increase.

An estimation of the protein in the cerebro-spinal fluid is of preeminent diagnostic importance. The protein in

the cerebro-spinal fluid in health consists chiefly of albumin and a smaller amount of globulin. Although the ratio of albumin to globulin changes in certain diseases of the central nervous system, the amount of globulin is never in excess of that of albumin. It is usual to measure both the amount of total protein and the increase of globulin. An increase in the value of total protein beyond 50 milligrammes per 100 cubic centimetres is regarded by the majority of pathologists as indicative of a pathological fluid. The total proteins are increased in all forms of meningitis, neurosyphilis, and generally in disseminated sclerosis, and may be increased in spinal and cerebral tumours, cerebral abscess, cerebral hæmorrhage and thrombosis. Globulin increase accompanies the increase of total protein, but the ratio varies in different diseases, as exemplified in the different colloidal reactions, the electrolytic dissociation in which being dependent upon the variation of this ratio.

Cell Protein Dissociation.

In some diseases the cells and protein show a corresponding increase; in others the values may be dissociated—protein increase without appreciable increase of cells (albumino-cytologic dissociation) or cell increase without appreciable increase of protein (cyto-albuminic dissociation). The former is the more important from a diagnostic standpoint. It is seen typically in the "compression syndrome." But it occurs also in some cases of cerebral tumour, localized meningitis, polyneuritis, myelitis and cerebral arteriosclerosis. In cases of spinal compression, albumino-cytologic dissociation is much more marked below the lesion than above it.

The Froin Syndrome.

The characteristics of the true Froin syndrome are: (i) Xanthochromia, (ii) massive coagulation (or great increase in total protein), (iii) pleocytosis, usually of the mononuclear type. The syndrome is practically pathognomonic of spinal compression, and the yellow colouring is due to venous stasis and stagnation of the cerebro-spinal fluid. It is seen in cases of chronic meningitis, especially of syphilitic origin, and in cases of transverse myelitis associated with a zone of meningitis, obstructing the spinal subarachnoid space. Likewise, the syndrome is found in cases of tumour of the cord or dura, spinal caries and epidural abscess. It is said to be a rare concomitant of Landry's paralysis and polyneuritis. These specific pathological changes occur typically below the level of the spinal compression, but recent work has shown that the same changes may be found directly above the level of the block, and Kamman reports such occurrence in extradural abscess and diffuse syphilitic meningo-myelitis. Cushing and Ayer have found fluid, presenting the appearance of a typical Froin syndrome, withdrawn from the spinal subarachnoid space immediately above tumours of the *cauda equina*.

It should be noted that the number and type of cells found in the Froin syndrome are subject to the widest variation, according to the nature of the lesion. The xanthochromia and excess of protein (or massive coagulation) may point to a condition of spinal block; it is upon the number and the nature of the associated cells that a specific diagnosis may rest.

Glucose.

Absolute agreement has not been reached with regard to the normal quantity of glucose in the cerebro-spinal fluid. The figures usually given as normal are 45 to 86 milligrammes per 100 cubic centimetres, and it is generally accepted that figures over 100 milligrammes per 100 cubic centimetres represent hyperglycorrhachia.

It has been established that the glucose content of the cerebro-spinal fluid varies with that of the blood sugar, although changes in the blood sugar are reflected more slowly in the cerebro-spinal fluid. Little reliance, therefore, can be placed on the estimation of glucose in the cerebro-spinal fluid, unless the blood sugar value is obtained at the same time or the estimation made after a fasting period. For this reason, although both increase and decrease of the cerebro-spinal fluid glucose occur in disease of the cerebral nervous system, its estimation is of slight diagnostic value, unless used in conjunction with other tests.

Hyperglycorrhachia.

Hyperglycorrhachia occurs characteristically in *diabetes mellitus*. It is said to have been noted also in other diseases, such as whooping cough, pneumonia, scarlet fever *et cetera*, being, however, in no way specifically related to the disease process, but to the hyperthermia associated with disease. An increase in the concentration of glucose has been claimed in acute epidemic encephalitis, multiple sclerosis and cerebral tumour; but such increase is apparently not constant and is not universally recognized as an important diagnostic aid.

Hypoglycorrhachia.

Hypoglycorrhachia is practically always associated with acute meningitis, whether of coccal or bacillary origin, the reduction being due to glycolysis. As the amount of protein present in a pathological fluid may prevent the reduction of Fehling's solution, it is necessary to remove the protein by precipitation before estimating the sugar. Sugar values are always below normal in tuberculous meningitis and in acute coccal meningitis, due, perhaps, to the presence of microorganisms and the enormous increase of polymorphonuclear leucocytes. Glucose may be quite absent from the cerebro-spinal fluid. This test, obviously, is of paramount significance in the examination of lumbar puncture fluid.

Chlorides.

The concentration of chlorides in blood and cerebro-spinal fluid offers a fairly constant ratio; and even though the mechanism of the transference of tissue chloride not obeying the ordinary laws of dialysis is not properly understood, variations of chloride concentration may be of diagnostic import. While not a definite rule, on account of anomalies sometimes found, high chloride values point to derangement of kidney function. This may clinch the diagnosis in case of coma of suspected uræmic origin. The cerebro-spinal fluid chloride varies with that of the blood, but never falls below it. Hence low values may be recorded in acute lobar pneumonia. Otherwise low chloride values are indicative of meningitis, and their determination is of distinct value in cases of cerebral abscess, cerebral tumour or encephalitis, where the cerebro-spinal fluid picture presented may, in all other respects, closely resemble that of meningitis.

The chloride value is not usually altered in neurosyphilis. Examinations made during the course of the disease show that progressively decreasing chloride values point to a fatal termination in meningococcal and pnuemococcal meningitis, and may thus be used prognostically.

Other Chemical Constituents.

The estimation of lactic acid does not usually come within the routine examination of the cerebro-spinal fluid, although there is general agreement that it is increased in cases of acute meningitis due to cellular metabolism, uræmia and eclampsia. It is also increased in epilepsy after convulsions. The percentage of urea in the cerebro-spinal fluid varies with that of the blood, and is of little or no value in the diagnosis of intracerebral conditions. There is some deviation in the percentage of calcium in cases of acute tetany, but its estimation has not proved of value in the investigation of neuro-psychiatric disorders. Uric acid, phosphates, nitrates, sulphates and magnesium have been found normally and abnormally in the cerebro-spinal fluid, but so far the values have not ascended to clinical significance.

The Colloidal Reactions.

Among the numerous colloidal reactions devised in these later years, the gold-sol test of Lange holds pride of place, being based on the pioneer work of Zsigmondy.

Gold in the colloidal state is precipitated by certain electrolytes, such as sodium chloride. It has been found that while albumin tends to prevent such precipitation, globulin tends to aid the process. In other words, in

certain pathological cerebro-spinal fluids the albumin exerts a protective action and the globulin manifests a precipitating action. Thus in syphilitic fluid the ratio of albumin to globulin is responsible for the precipitation of the gold in an extended series of dilutions. Various degrees of precipitation are manifest by changes in colour. This may be expressed either in figures or by means of a curve. In a general way, fluids of paretics cause precipitation in the lower dilutions, those of tabetics in the medium dilutions, while the fluids from patients suffering from meningitis cause precipitation in the higher dilutions only. A typical paretic response would be expressed thus: 5554432100, a tabetic as 0014320000, and a meningitic as 0000123320.

The albumin-globulin ratio in pathological fluid which is assumed to be the cause of colloidal precipitation (and is seen typically in cases of neurosyphilis), is not the agent responsible for causing deviation of complement in the Wassermann test. Paretic and luetic curves have been obtained from fluids which persistently fail to react to the Wassermann test, as, for instance, in cases of disseminated sclerosis and epidemic encephalitis and even in other pathological fluids with an altered protein ratio. It must not therefore be assumed that any colloidal test is specific, but such test may be of ancillary value in diagnosis in the presence of either a positive response or a failure to react to the Wassermann test.

Many clinicians still aim at being able to draw a precise line of demarcation between various types of neurosyphilis, and they rely with a certain pride upon the colloidal reactions to establish a diagnosis of paresis or meningovascular syphilis, as the case may be. Acccepting, as I do, the "unity-in-variety" concept of neurosyphilis, from an experience of several hundred cases, I have come to regard the colloidal reactions as indicating the severity of the syphilitic involvement rather than pointing to a definite clinical type. And I have found a change in colloidal reaction from the "paretic" to the "meningitic" zone to follow a period of successful treatment. The Lange gold-sol test may sometimes, therefore, find its most useful place in prognosis, when the curve changes from what we may call a malignant to one of more benign type.

Other colloidal tests have from time to time been investigated, the chief being the mastic, benzoin and paraffin reactions. Quite recently Takata and Ara introduced a very much simpler one-tube colloidal test, which experience has proved to be of value in suspected syphilitic fluids, and de Thurzo has advanced the claims of a bicoloric colloidal reaction which makes for greater facility in differentiation of the various zones. But none of these tests seem to have displaced the original gold-sol test of Lange. Some, I think, are easier to perform, and the manufacture of suitable colloidal gold can be very exasperating at times; some appear to be less subject to subtle changes in reaction and more stable on keeping, but none give the fine precision, the nice distinction of colour variation, and none appear more accurate than the colloidal gold.

The Wassermann Test.

Kolmer, from his extensive immunological studies, concludes that normal cerebro-spinal fluid does not contain the normal antibodies found in the blood, presumably because of a selective inhibitory mechanism of the cells concerned in the secretion or excretion of the cerebro-spinal fluid. And, further, that it is probable that the Wassermann and other antibody-like substances of syphilis are produced in the meninges, brain and spinal cord as reactionary substances of cells in contact with the *Spirochæta pallida*. For this reason the cerebro-spinal fluid may yield a positive Wassermann reaction while the serum fails to react.

Although the exact basis of the reaction remains obscure, the Wassermann reaction is at present the most important biological test in the diagnosis of neurosyphilis. "False positives" are said to occur in highly albuminous yet non-syphilitic fluid, but this may be largely overcome by inactivating the fluid.

A strong and consistent positive Wassermann reaction is the strongest evidence of a spirochætal involvement of the central nervous system. A failure to react is of somewhat doubtful value, because certain types of neurosyphilis fail to give the positive response. Experience proves that in about 20% of tabetics and in a somewhat larger percentage of syphilitic hemiplegics, the cerebro-spinal fluid fails to react to the Wassermann test. Apart, however, from deficiencies in technique with highly albuminous fluids and a few rare cases of meningitis where results are paradoxical, the cerebro-spinal fluid yields a positive Wassermann response only in cases of neurosyphilis and such conditions (rare in this country) as sleeping sickness and leprosy. A positive result is obtained in a certain percentage of syphilitics in the late primary and secondary stages, and in cases which exhibit no definite clinical signs (*paresis sine paresi*). In congenital syphilitics, the results are variable; the greater number give positive reactions in childhood and tend to fail to react with advancing age. The response in cerebral gummata is inconstant, presumably depending upon the presence or absence of meningitic involvement. The response varies also in accordance with previous treatment.

In general, a positive Wassermann response in an organic nervous lesion serves to establish a specific pathological foundation. Syphilis can be diagnosed in an organic nervous disorder yielding no reaction to the Wassermann test only by the aid of collateral tests. And these, namely, cytological investigation, protein determination and colloidal reaction, will, in most cases, adequately establish the diagnostic trend.

Of the various modifications of the Wassermann test and the flake precipitation reactions, little need be said, The literature is conflicting and dogmatic pronouncement is impossible in the absence of an extensive series of comparisons. While each may claim different advantages in technique, the consensus of opinion is that they are in no way more sensitive than the Wassermann test.

Conclusion.

Lumbar puncture, which includes the manometric study of cerebro-spinal fluid pressure and the subsequent physical, chemical and bacteriological examination of the fluid is without doubt the most important diagnostic aid in all cases of organic disease of the nervous system. It is true that the results of such examinations are sometimes equivocal or incapable of exact interpretation. It is true also that many specimens of fluid yield results which are all within normal limits; that in the heredodegenerations, such as Huntington's chorea and amyotrophic lateral sclerosis, examination of the fluid gives no clue to the underlying pathological change. And it is equally true that lumbar puncture is frequently barren of interest in the biogenetic psychoses and amentias. But even in the present incomplete state of our knowledge, both of the mechanisms of many reactions and their significance, examination of the cerebro-spinal fluid may supply evidence of inestimable diagnostic value.

The interpretation of the findings, as well as their correlation with the clinical signs and symptoms, presents in a number of cases the greatest difficulty, because in the majority of instances the results of cerebro-spinal fluid examinations are not of themselves pathognomonic of any one specific condition. But, rightly interpreted, they may turn the diagnostic weathercock in the proper direction.

In general practice, I believe, lumbar puncture is not performed as often as its value would seem to indicate, and this may be due to the alleged risks involved in the procedure. The risks, however, are generally trifling in comparison with the knowledge to be gained. The greatest risk lies in a badly performed puncture in the case of a patient with a suspected cerebral tumour, or more pertinently, where there is clinical evidence of an expanding lesion beneath the tentorium. In such cases, where there are obvious signs of tumour with marked papillœdema, it were perhaps best not to carry out a lumbar puncture, the value of which would be merely confirmatory. But where the clinical signs are not so obvious, and where such conditions as sinus thrombosis, cerebral abscess or neurosyphilis have entered into the arena of differential diagnosis, lumbar puncture becomes an almost vital necessity. Carried out with due deference to its risk, with

the patient in the horizontal position, with a needle of small calibre, only the smallest quantity of fluid being withdrawn, and the patient kept recumbent for the ensuing twenty-four hours, the operation may be said to be fairly free from danger.

Many aspects of diagnostic lumbar puncture have not been touched upon in this paper. Many special tests have not been mentioned. But, conscious of my shortcomings in this and other respects, I have endeavoured to present the broader outlines of the subject and to leave the details, especially where of a contentious or inconclusive nature, to be followed up by those who care, in the now extensive literature upon the cerebro-spinal fluid.

We are still but a little way beyond the threshold of knowledge concerning the physiology of the cerebro-spinal fluid, and its reaction to disease of the central nervous system remains deep in the shadow of our ignorance. The lumbar puncture needle, however, is the neurologist's divining rod. Though its use may be limited, its value can never be belittled. Without its aid the diagnosis of many intracranial conditions would amount to little more than neurological necromancy.

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Hospitals.

ROYAL COMMISSION ON THE HOBART PUBLIC HOSPITAL.

THE Royal Commission on the Hobart Public Hospital has made its report. The commissioners were Mr. Edward Laret Hall, Police Magistrate; Dr. Eric Cooper, Medical Superintendent of the Melbourne Hospital; and Mr. Dudley Keith Otton, Secretary and Accountant of the Hospital Commission of New South Wales.

The terms of reference of the Commission were as follows:

(i) To inquire into and report upon the irregularities at the Hobart Public Hospital reported upon by the Auditor-General of the State of Tasmania to the Board of Management of the said Hospital in December one thousand nine hundred and twenty-eight and in each subsequent year.

(ii) And to inquire specifically and to report whether such irregularities or any of them are due to the failure of the said Board and/or its officers and servants or any of them to efficiently and properly discharge their duties according to the *Hospitals Act* 1918 and the rules and regulations of the Hospital approved by the Governor on the sixteenth day of February one thousand nine hundred and twenty and any amendments thereof.

(iii) To inquire generally into and report upon the administration management and control of the said Hospital and all matters and things connected therewith and incidental thereto and to make such recommendations as you may think fit for the improvement thereof and for the better discovery of the truth in the premises.

The Commissioners summarize their conclusions and recommendations in the following terms:

(1) The responsibility for the loss of 21 milligrammes of radium, valued at approximately £250, rests upon the Board of Management, the Surgeon Superintendent, and the Senior House Surgeon. More care and a proper system of control of radium would have prevented these losses, and is recommended.

(2) The discrepancies in stores and equipment, amounting to approximately £2,182, are due to the failure of the Board of Management and its officers to instal and carry out a proper system of stores and stock control. The shortages consist of actual losses by theft or carelessness, and goods which may have been used in the Hospital but cannot be accounted for.

The immediate installation of the ward system, as advised by the Audit Department, is recommended to prevent further losses.

(3) The Board of Management has failed to realize its responsibilities in controlling the expenditure of public funds, and has shown apathy, indifference, and often passive resistance to recommendations and advice from authorities. The Board has failed to supervise the work of its staff, and to take action when it was apparent that losses were occurring owing to the inefficiency and incapacity of senior officers. A change is recommended from the nominee system to one of control by a Board of nine, three to be appointed by the Governor in Council and six elected by the subscribers to the Hospital.

(4) The sub-committees of the Board have not carried out their duties in a satisfactory manner. The appointment of expert committees to advise and control the Hospital officers in certain directions is recommended.

(5) For some time prior to his resignation the former Secretary, owing to age and infirmity, was incapable of efficiently carrying out his duties. Many of the troubles of the Hospital are attributable to his deficiencies. The appointment of a chief executive officer with the necessary qualifications and business ability to manage the affairs of the Hospital is advised.

(6) The Surgeon Superintendent is not a good general administrator, and his interests lie more in the practice of surgery than in the detailed supervision and management of the Hospital. As the expert adviser of the Board he must accept responsibility for its failures in many directions.

It is recommended that the position of Surgeon Superintendent be abolished, and a Medical Superintendent appointed to direct the medical services of the Hospital, the administration of the institution to be mainly in the hands of the Secretary.

(7) The former Matron, by reason of age and disability, was unable to keep control of the detailed working of the wards, nor was she able to assimilate new methods. The discrepancies in stores and equipment are in a large part due to her incapacity. The present Matron should be encouraged to improve the discipline and standard of the nursing services, and should report direct to the House Committee, and should not be under the charge of the Surgeon Superintendent.

(8) The nursing staff is insufficient in numbers—their hours are too long, and the addition of at least ten extra nurses, with the provision of increased accommodation, is necessary.

The present antiquated and uncomfortable uniform should be replaced by a more simple, modern, and sanitary outfit.

(9) The honorary medical staff should be developed—beds being allotted to each member of the staff.

More honorary anæsthetists should be appointed.

(10) A medical radiologist and a pathologist should be appointed on a part-time basis.

(11) The work of the resident medical officers should be made more instructive; more surgical work should be given to them, and they should keep the medical records of the patients.

The number of junior residents should be increased to four.

(12) The food services of the Hospital are unsatisfactory and extravagant. One centralized kitchen with modern plant, and the appointment of a trained dietitian, are advised.

(13) The expenditure on provisions is excessive, and the ration scheme as suggested by the Audit Department should be established in its entirety.

(14) The installation of a stock system in the dispensary and improved methods of purchase of supplies, with the appointment of an expert Drug Committee, are recommended.

(15) The expenditure on surgery and dispensary supplies is excessive—outstanding items being surgical instruments and appliances and the remarkable consumption of alcohol.

(16) Standardization and economy in printing and stationery is advised. The publication of an annual report of the work, statistics, and financial statement of the Hospital is recommended.

(17) Standardization of stores and equipment, with bulk purchase of some items, such as linen, crockery *et cetera*,

and the calling of competitive tenders for others, with direct purchase and variable contracts for consumable commodities, is recommended.

(18) The provision of a laundry at the Hospital is essential, but under present financial conditions the modernization of the laundry at the New Town Infirmary appears to be more advisable.

(19) Appalling extravagance exists in the consumption of fuel and light. The capital cost of reorganization of the engineering services would soon be returned, as the annual saving is estimated as at least 25 per cent. of the outlay. Some means should be found to finance such a remunerative investment. A new incinerator should be provided.

(20) Present buildings are mostly old, scattered, and ill-adapted to the requirements of a modern hospital. A general building plan for the future, when money is available, should now be prepared, and any alterations or additions should conform to it.

(21) The statistics relating to number of patients, stay in days, attendances, and mortality are valueless for comparative purposes, and should be reorganized immediately on an approved system. The medical records are unworthy of the Hospital, particularly in the out-patient department.

(22) Many Hospital beds are occupied unnecessarily—the use of convalescent and chronic hospitals should be extended.

(23) The number of complaints from dissatisfied patients indicates that the attention received by patients is not always what it should be. Suggestions are made for the improvement of conditions at the Hospital.

(24) The out-patient department should be open daily, and staffed by honorary medical officers. The overlap of activities of the Charitable Grants Department and the Hospital should be remedied.

(25) The extension of a district nursing scheme is advised as an adjunct to the work of the Hospital.

(26) The appointment of a qualified almoner to co-ordinate charitable work, control the financial collections, from patients, and, in general, improve the service of the Hospital to the community, is recommended.

(27) The method of assessment and collection of fees needs revision, and this could be best effected by the appointment of an almoner.

(28) The classification of patients into public, intermediate, and private is necessary to bring the Hospital into line with modern ideas.

(29) Up to the present the Hospital has been largely supported by the Government; owing to financial stringency income from voluntary sources must be increased by intensive organization of public support.

(30) A contributory scheme bringing a flow of money to the Hospital and giving benefits to the members should be established.

(31) Changes in the method of allocation of Government subsidy are advised, with the establishment of a Hospitals and Charities Advisory Board to assist the Minister.

(32) An extension of the ladies' auxiliary scheme should be undertaken.

(33) The system of book-keeping and accounts does not conform to modern requirements, and should be reorganized as part of a uniform system for all the subsidized hospitals of the State, and in the Hobart Hospital should include a costing system.

(34) An itemized budget should be prepared at the beginning of the financial year, and utilized as a means of controlling expenditure.

(35) Amendments of the *Hospitals Act*, 1918, are necessary to give effect to many of the recommendations contained in this report.

Correspondence.

THE WORSHIP OF THE TEST TUBE.

SIR: May I sound, through your columns, a note of warning in the ears of those who are responsible for the training of medical students, more particularly to those worthy educators in the State of Victoria, where the medical curriculum is about to undergo another of its many metamorphoses?

The development of medical science was a very gradual process indeed before the days of Pasteur, when the rôle of bacteria was placed in its proper relation to disease. Now, within half a century or so, the mass of medical knowledge has assumed such vast proportions that no one man can practise competently in all its branches. As a result the medical curriculum has become the subject of almost constant controversy, and the medical student is forced to conform to a general more or less adequate academic programme embracing the basic sciences which, in the later years, becomes decorated by a variety of special subjects conditioned largely by the private enthusiasms of a handful of scientific idealists.

The Book of Life, we are told, begins with a man and a woman in the Garden, and ends with Revelation. But the modern medical student is more scientific. Those responsible for his training have decreed that he shall study medicine rather than man, and so he is introduced to life in the amœba and his biological investigations are fulfilled in the frog. True, he pores intently over the structural complexities of the cadaver and diligently attends the physiological demonstrations on the dog. He is made happily conversant with every appearance of diseased tissue and learns to utilize the empiricism of the British Pharmacopœia. But he is taught little or nothing about individual behaviour, either in health or disease. Therefore, he sees the diseased organ rather than the diseased man, and if no one particular organ is palpably diseased, he hunts round for the trothless tonsil or fixes his rapt attention to a carious tooth.

It is fashionable to smile at the old-fashioned doctor of former days, with his courtly gestures and his bizarre beliefs, to look tolerantly upon the family physician who knew not the ways of Wassermann and whose only diagnostic aids were his five special senses. But the old gentleman often gained more insight into the behaviour problems and minor maladies of his patients than the modern clinician does with all his electrocardiographs, ophthalmoscopes, cystoscopes and sphygnomanometers, and a biochemical laboratory at his elbow. His training was meagre because there was little to teach. His knowledge of physiology was lamentable. He gained his experience at first hand and did not have laboriously to learn to do a great number of things which he would never be called upon to do, and in consequence he did not have to sit down in his surgery waiting for patients to present themselves with the illnesses which he had been taught to understand. Within his obvious limitations he saw his patient as a human being, not as something in a test tube. He saw him as a more or less complete biological unit brought low by sickness or at war with his environment. His patient was a human being, and not an enlarged prostate or prolapsed uterus, or even a swarm of septic foci.

Nothing is further from my intention than to belittle or decry the truly scientific side of medicine. I am fully cognizant of the inestimable benefits to humanity which have resulted from the application of science to the healing art. But it is none the less true that the present teaching of medicine allows the student to become blinded to the proper understanding of human conduct in the bright glare that emanates from a variety of spectacular scientific procedures, and appears to foster a respect for the test tube out of all proportion to its value. So that there are now many practitioners who, confronted with a tube of urine, can see in its expert analysis a correct reflection of renal dysfunction, but whose vision fails to encompass the illness of the individual as a whole.

Our present teaching tends to over-emphasize the rarer diseases at the expense of the commoner complaints. It fosters too great a dependence on the ancillary physical and biochemical aids to diagnosis and pays too little attention to the conduct disorders and "functional" ailments which so constantly occur in private practice. In neurology, for instance, about 90% of the teaching is directed to organic neurological conditions, the majority of which are rare, and about 10% to the "neuroses" which are common. So that the present-day graduate will diagnose the occasional pontine tumour or Fröhlich's disease and

overlook or misunderstand the far more frequently seen anxiety neurosis. And that is why a fractional test meal is carried out on the neurotic who complains of flatulence, and the appendix is removed from the damsel whose conversion hysteria has directed the surgeon's palpating finger to the right iliac fossa. The present graduate is better equipped to remove a gall-bladder than to unearth an intrapsychic conflict, so the gall-bladder goes the way of all flesh and conflict finds fresh expression in another organ.

The worship of the test tube is widespread. There is not a fluid in the whole human body, be it excreted or secreted, eliminated or ejaculated, that is not sacrificed to the test tube. The simple neurotic, and his name is legion, is the richest patron of the test tube worshippers, and when he is tested and measured and operated upon, and finally dosed with potassium bromide by our present learned graduates, he falls into the hands of the quacks with a want of confidence or a frank contempt for the medical profession, which has taken his money for tampering with a condition it has never adequately been taught to understand.

Let the test tube remain and all that glittering array of implements that goes with it, but in teaching the student the value of these things, do not allow him to become medically myopic. Let the future practitioner be a man dealing with men, not a trained test tube worshipper treating a laboratory animal. Teach him to rely upon his own insight first and on the trained laboratory assistant afterwards. Teach him to evaluate the interrelation of physical and intellectual factors in his patients and not to lose sight of the individual in the illness. In other words, teach him to see the body as a whole.

Yours, etc.,

REG. S. ELLERY, M.D.

14, Collins Street,
Melbourne,
November 2, 1931.

"AVERTIN."

SIR: I should like you to publish the following reaction to "Avertin" anæsthesia, with the object of obtaining an explanation of: (i) an unusual precipitation during the preparation of the solution, (ii) an almost tragic collapse during anæsthesia.

The patient, a female, aged twenty-three years, weight eight stone one pound, healthy, of normal type, and rather highly strung than otherwise, was submitted to operation on October 5, 1931, for curettement and section, "Avertin" being chosen as the anæsthetic. Basal anæsthesia of 0·1 gramme per kilogram in 2·5% solution was used. The "Avertin" was added to distilled water at 100° F. slowly, the solution being constantly stirred. A very small amount of white solid was precipitated and did not redissolve. It was noted that the water had now cooled to 90°, and the container was reimmersed in water at 100°. The precipitate persisted, and discarding of the solution was considered, but the Congo red test failed to show dissociation. In view of this and the small amount of precipitate, it was decided to continue, and the anæsthetic was administered in the usual way, the patient being moved to the theatre after twenty minutes, in a state of normal "Avertin" anæsthesia. A small amount of ether (open method) was required during dilatation of the cervix. About fifteen minutes after opening the abdomen the anæsthetist told me the patient's condition was becoming bad. A little over one ounce of ether had been used. Within a few minutes the breathing practically ceased and was only observable by the slight epigastric movement. At the same time the pulse failed completely at both wrists. A slow, steady heart beat of sixty to seventy per minute was heard with the stethoscope. The colour was deathly white, there having been no cyanosis at any stage. One cubic centimetre of pituitrin was given and the operation hurriedly continued and completed in five to seven minutes. The patient was returned to bed immediately, warmth applied, adrenalin, 15 minims, given hypodermically, the bowel washed out, and a pint of glucose saline given rectally. The condition was then as above. After a quarter of an hour the pulse was just palpable at the wrist, but for the next two hours was very weak, the respirations scarcely perceptible, and the colour fearfully pale. Steady improvement then set in, and in an hour all danger was passed.

I would add: (i) Premedication consisted of atropine, one one-hundred-and-fiftieth of a grain, without any morphine. (ii) The patient was not of the subthyreoid type. (iii) No thyroxin was available. (iv) The "Avertin" was fresh, and anæsthesia during the preceding week with the same solution was perfect and without worry.

If any with more experience than I in "Avertin" anæsthesia will offer an explanation of the behaviour of the "Avertin" in preparation and comment upon the collapse, I shall be grateful. This experience is so contrary to the usual reports of "Avertin" anæsthesia that it seems worth discussing.

Yours, etc.,

H. W. HORN, M.B., Ch.M.

Brisbane,
November 13, 1931.

THE CUTANEOUS TUBERCULIN TESTS OF PIRQUET AND MANTOUX.

SIR: Parodi, in *La Presse Médicale*, number 50, of June 21, 1930, has a long article on the interpretation, physiological, pathological and clinical, of the cutaneous allergy of Pirquet in pulmonary tuberculosis. His conclusions (theoretical) are:

(i) It may be doubted if there exists a veritable hypersensibility of the organism to the toxins of tuberculosis; in all cases the allergy of Pirquet is not a humoral reaction; it is not a reaction of immunity.

(ii) The cutaneous reactions to tuberculin are of a trophic reflex nature.

(iii) As a matter of fact, the cutaneous reactions are not specific; they are local and closely related to other motor trophic and sensory reflexes. The reactions are influenced by physical and physiological factors and by the general tonus of the neuro-vegetative system.

Olshausen, of Leipzig (*La Presse Médicale*, number 59, July 25, 1928) says that Keller has been able to produce among the tubercular, by means of a concentrated glycerinated bouillon containing neither tubercle bacilli nor tuberculin, the focal and general reactions considered as characteristic of tuberculin, and that it is impossible to distinguish specific reactions in a tubercular subject.

J. Troisier and M. Monnerot-Dumaine (*Revue de la Tuberculose*, Volume XI, number 4, April, 1930) state that the cuti-reaction in man produced by concentrated bouillon-glycerine can be identical in its evolution with that produced by tuberculin.

Yours, etc.,

J. MORRIS ROE.

Victory Chambers,
Queen Street,
Brisbane,
November 20, 1931.

RADIOLOGICAL EXAMINATION OF THE HEART.

SIR: The review in the last number of THE MEDICAL JOURNAL OF AUSTRALIA (October 17, 1931) of the address of G. Grant Allan to the Royal Society of Medicine has special interest for radiologists in Australia because of the statement that Australian medical practitioners do not make use of radiology to assist in the diagnosis of cardiac lesions.

During a recent visit to England in my association with Dr. Peter Kerley at the Royal Chest Hospital in London, it was my privilege to "sit at his feet" for some months in the radiological department at that hospital. The facility and uncanny accuracy with which he diagnosed cardiac conditions from an X ray film alone without any history of cases were a source of wonder and an inspiration

to study these methods. Mitral stenosis, enlargement of the left ventricle associated with hyperpiesis, aortic insufficiency, and the heart outline in the later months of pregnancy were a few of the conditions on which he made accurate comments. There was no need to make the intricate measurements of detailed cardiometric methods, for his intuition was the result of a past critical study of these.

The essentials of his technique were high milliampèrage, rapid exposure, and two metre distance from target to patient. A very sharp and clear cardiac outline can thus be obtained without distortion, as the central rays only are absorbed by the heart tissue, and the rapidity of exposure—one-twentieth to one-tenth of a second—obviates blurring of the outline by the movements of the heart. Orthodiagraphy, on the other hand, is a time-consuming and very clumsy method of making heart measurements, and in London is being superseded by the "snap" film at two metres.

Dr. Grant Allan's paper should give a stimulus to this method of diagnosis, which is a powerful link in the chain of evidence to fix a diagnosis in cardiac conditions, both primary and secondary.

Yours, etc.,

KEITH H. HALLAM.

Radiological Department,
Saint George's Hospital,
Kew,
Victoria,
October 23, 1931.

TRAUMA AND ORGANIC VISCERAL DISEASE.

SIR: In a recent paper (THE MEDICAL JOURNAL OF AUSTRALIA, November 7) I drew attention to the fact that in more than one case, one of them being *Ryman versus Municipal Council of Mosman*, medical witnesses had asserted in a workers' compensation court that muscular effort increased peristaltic action. I wrote a little more, but this was deleted in the final edition and not printed. As it turns out, the deletion was a mistake. Readers will note the paragraph beginning: "Now, if it is true that muscular effort causes increased peristalsis, these gentlemen, all of them, have found something very far-reaching indeed." Then attention is called to the fact that if it is true, it would enable us to explain appendicitis as due to the work a man was doing. Next came the deleted paragraph, which was indeed actually read at the meeting. It was as follows:

> Still, would it not be better for men who have made new and important discoveries in physiology to publish them, with all the equally important evidence, in some great scientific journal? Instead of this, they say nothing about it, except to a few laymen in a workers' compensation court, and the world is deprived of their message. If this is modesty, it is too much modesty, it is modesty that actually becomes a fault.

Now we have Dr. F. S. Stuckey, in your issue of November 21, writing and taking up a most amazing position. He says he wishes to protest at the "gibe" at the opinions of other medical men "without producing any evidence to show their falsity." That is, I am not permitted to refuse to accept his bald *ex cathedra* deliverances as opinions given without evidence. He thinks he is entitled to claim from me "evidence to show their falsity." Such is his sublime confidence in his own infallibility and his belief in his right to special privilege.

What readers of THE MEDICAL JOURNAL OF AUSTRALIA will ask, as reasonable men, is what I ask. If Dr. Stuckey has found out a new fact in physiology, let him write a serious scientific paper and tell us all about it. He has invited the readers of the journal to join in a discussion on the subject of internal hernia. Good. But the proper way to begin the discussion will be for Dr. Stuckey to commit his ideas seriously to paper about this too. But let him tell us first of all about the causation of increased peristalsis by muscular effort.

Carried away by the childlike and uncritical respect given to their orders and foibles and opinions by patients, many medical men seem in time to take themselves as seriously as their patients do. They come to regard themselves as really oracles, they conceive their opinions to be no ordinary opinions, like those of other men, but as inspired truth, to question which is profanation. They have not practised the wholesome habit of standing apart, in imagination, from themselves, and laughing at themselves. And so, when some vulgarly irreverent person calls their opinions bunk, and laughs, they feel surprised and pained and annoyed.

Is Dr. Stuckey one of these humourless people? Let us have those two papers.

Yours, etc.,

C. E. CORLETTE.

Sydney,
November 24, 1931.

SIR: Dr. C. E. Corlette's articles on visceral traumata in your issue of November 7 are characteristically stimulating. The most intriguing problem discussed is that of the relationship of muscular effort to intraabdominal catastrophes. The physical conditions in the abdomen during effort which the author postulates, would appear to be too simple, and the effects of the forces concerned more elaborate than he states them to be.

First, the abdominal contents consist of a discontinuous medley of semi-fixed solids, of semi-solids, fluids and gases. Hydrostatic conditions are predominant, but subject to certain limitations owing to discontinuities and restrictions to free movements. Boyle's law, which relates to gaseous states, cannot have any general application to intra-abdominal conditions.

Secondly, the articles point out that an increase of intraabdominal pressure involves a corresponding increase of pressure within a hollow viscus. The converse proposition is not admitted. An increase of intravisceral pressure, represented by the filling of the stomach with fluid, is considered to be not compensated by the general intra-abdominal pressure. This would be the case if the additional weight were supported by the attachments of the stomach, but this possibility is denied by the author, who insists that the stomach is pressed constantly against the liver and is never strained away from it. One of these two alternatives must be conceded or a new factor admitted throughout, that of the elasticity of the viscus itself.

Thirdly, the operation of static forces only is postulated by the author in regard to this effort problem. Dynamic forces must, however, appear whenever the shape or volume of the abdominal cavity is altered. At least two possibilities of such an occurrence present themselves. A strong contraction of the abdominal muscles, with the glottis closed, will cause some alteration of shape of the abdominal cavity and some relative movement of abdominal contents. A more important shearing stress will result if an expiration be made during strong effort. The diaphragm in this case will move suddenly upwards and work will be done on the abdominal contents in restoring the equilibrium of pressure which has been disturbed. Differential stresses will be in evidence particularly at the initiation of these relative movements and in the checking of them by limiting tensions or pressures. If the effort be severe and the disturbance of equilibrium sufficiently sudden, it is conceivable that the forces which result could be sufficient to disrupt diseased tissue exposed to strain, or effect an internal herniation.

It would appear, therefore, that there is at least a possibility of muscular effort being causally related to a catastrophe, such as the rupture of a gastric ulcer.

Yours, etc.,

H. K. FRY.

Adelaide,
November 25, 1931.

Post-Graduate Work.

LECTURES IN MELBOURNE.

THE Melbourne Permanent Post-Graduate Committee has announced the titles of the post-graduate lectures to be delivered in Melbourne by Mr. C. H. Fagge. The lectures will deal with acute abdominal emergencies as follows:

1. General points. Appendicitis.
2. Gynæcological emergencies.
3. Obstruction.

The lectures will be delivered in the Medical Society Hall at 8.30 p.m. on February 11, 12 and 15, 1932.

Diary for the Month.

DEC. 15.—New South Wales Branch, B.M.A.: Medical Politics Committee.
DEC 18.—Queensland Branch, B.M.A.: Council.

Medical Appointments.

The undermentioned have been appointed Honorary Anæsthetists at the Adelaide Hospital, South Australia: Dr. K. F. Cooper, Dr. H. M. Fisher (B.M.A.), Dr. O. W. Frewin (B.M.A.), Dr. R. L. Kenihan (B.M.A.), Dr. H. G. Prest (B.M.A.) and Dr. N. S. Gunning.

* * *

Dr. A. I. Chapman (B.M.A.) has been appointed Certifying Medical Practitioner at Minyip, Victoria, pursuant to the provisions of the *Workers' Compensation Act*, 1928.

* * *

Dr. E. Mansfield (B.M.A.) has been appointed as Quarantine Officer at Cairns, Queensland, pursuant to the provisions of the *Quarantine Act*, 1908-1924.

* * *

Dr. A. C. Herrington (B.M.A.) has been appointed Government Medical Officer at Bowral, New South Wales.

* * *

Dr. N. A. D. Keirle (B.M.A.) has been appointed Government Medical Officer at Cumnock, New South Wales.

* * *

Dr. C. J. M. Walters (B.M.A.) has been appointed Senior Medical Officer, Coast Hospital, Office of the Director-General of Public Health, New South Wales.

* * *

Dr. T. Black (B.M.A.) has been appointed Medical Officer, Department of Mental Hospitals, New South Wales.

* * *

Dr. C. E. Phillips has been appointed Medical Officer, Department of Mental Hospitals, New South Wales.

* * *

Dr. W. E. Hasker (B.M.A.) has been appointed Acting Government Medical Officer, Dalby, and Acting Visiting Medical Officer to the Jubilee Sanatorium, Dalby, Queensland.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xvi.

DALWOOD HEALTH HOME, BALGOWLAH, NEW SOUTH WALES: Staff appointments.
INSPECTOR-GENERAL OF HOSPITALS DEPARTMENT, ADELAIDE, SOUTH AUSTRALIA: Honorary Clinical Assistant.
PERTH HOSPITAL, PERTH, WESTERN AUSTRALIA: Junior Resident Medical Officers.
ROCKHAMPTON HOSPITALS BOARD, QUEENSLAND: Resident Medical Officer.
ROYAL PRINCE ALFRED HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Vacancies.
TIBOOBURRA DISTRICT HOSPITAL, NEW SOUTH WALES: Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members desiring to accept appointment in ANY COUNTRY HOSPITAL, are advised to submit a copy of their agreement to the Council before signing, in their own interests. Brisbane Associated Friendly Societies' Medical Institute. Mount Isa Mines. Toowoomba Associated Friendly Societies' Medical Institute.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

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